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THE
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Vol. 21, No. 12 December, 1955

MECKEL'S DIVERTICULUM: A REPORT OF NINETY-THREE CASES

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Meckel's diverticulum is not a rare anomaly; an incidence of 1 to 2 per cent is reported in various autopsy series.[4,8] Its presence becomes of importance only when complications arise. The patients who were operated upon constitute a very small proportion of the total, so that the number of patients coming to operation is limited.

We are reporting 93 cases of patients with Meckel's diverticula, who were operated upon primarily for intrinsic pathologic changes resulting from the diverticula, or removed incidentally during other surgical procedures, with 22 of the series being found at necropsy in patients who died from other causes (tables 1 and 2).

These 93 cases were noted in 86,000 intra-abdominal operations done at the Lahey Clinic, an incidence of 1.1 per 1000, which is much lower than the incidence noted at necropsy. Obviously, many Meckel's diverticula are present and remain undiscovered during intra-abdominal surgical procedures. It is evident that in some of these patients, serious complications may develop later that could well have been avoided if the diverticula were found and excised when the abdomen was opened for other surgical procedures. Noel[12] reported 25 cases in 1200 intra-abdominal operations, or an incidence of 21 per cent.

Meckel's diverticula as reported in the literature occurs more frequently in the male, being reported as 2 to 4 times more frequent than in the female; however, 53 of this group of 93 patients were male.

Although several authors had described the anomaly previously, it was not until Meckel's series of articles in 1808 and 1812 that the relationship to the omphalomesenteric duct was clearly shown, and that the diverticulum was the result of persistence of a portion of this duct. Cohen[5] noted that Fabricius described an intestinal diverticulum in 1598, and Curd[6] quoted a reference to a diverticulum of the terminal ileum by Lavater in 1672. Ruysch in 1701 published

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TABLE 1

Diagnosis of intra-abdominal pathology—51 cases

(Meckel's diverticulum was incidental finding at operation)

Diagnosis	Number
Cholecystitis	4
Common bile duct stone	1
Duodenal ulcer	2
Carcinoma of duodenum	1
Acute pancreatitis	1
Splenic cyst	1
Intestinal obstruction	2
Regional ileitis	6
Leiomyoma of ileum	1
Appendicitis	9
Ulcerative colitis	4
Carcinoma of colon	4
Diverticula, colon	1
Ventral hernia	1
Inguinal hernia	1
Femoral hernia	1
Prostate	1
Myomata uteri	9
Ovarian cyst	1
Total	51

TABLE 2

Necropsy material in which Meckel's diverticulum was an incidental finding

Principal Cause of Death	Number
Hypertension	2
Adrenal tuberculosis	1
Brain tumor	5
Carcinoma of esophagus	1
Common bile duct stricture	2
Duodenal ulcer	1
Carcinoma, head of pancreas	1
Subarachnoid hemorrhage	1
Cranial aneurysm	1
Hydrocephalus	1
Cardiac	4
Carcinoma, lung	2
Total	22

the first illustration of a Meckel's diverticulum. Littré noted the presence of such a diverticulum in an umbilical hernia in 1700. Zenker, in 1861, found aberrant pancreatic tissue in the diverticulum. Gastric mucosa was noted in the lumen and its relationship to hemorrhage was established by Denecke,[7] in 1902.

EMBRYOLOGY

The omphalomesenteric duct forms a normal communication between the primitive midgut and the yolk sac which narrows gradually and finally disappears at the 7 mm. stage of embryonic development. All or a portion of this structure may persist, but it is the ileal segment that forms the diverticulum that bears Meckel's name. Other anomalies may include a patent vitello-intestinal duct, omphalomesenteric cyst, and umbilical sinus. Any of these also may possess vestigial bands, which can provide a means of volvulus of the intestine around it with resultant obstruction and strangulation. Meckel's diverticulum should fulfill the following criteria: (1) it should be situated on, or near, the antimesenteric border of the ileum, (2) it must have its own blood supply, and (3) it must contain all the layers of the bowel.

ANATOMY

Size. The average size of Meckel's diverticulum is about 5 cm. in length with the communication with the ileum varying from 1 to 7 cm. in diameter. Several longer diverticula have been described, one up to 96 cm. by Chaffin,[3] but it is the opinion of most authors that those diverticula over 25 cm. belong to the classification of ileal reduplications.

Location. Jay and co-workers[9] noted that 75 per cent of all Meckel's diverticula are within 36 inches of the ileocecal valve, but add that they found one 66 inches from the ileocecal junction. Any exploration for Meckel's diverticula should include sufficient examination of the bowel to encompass all of these possibilities, no matter how remote, since in all of those patients in whom this is undertaken, the diagnosis may be obscure and a possible Meckel's diverticulum should not be overlooked by a cursory examination.

PATHOLOGY

Pathologic complications of Meckel's diverticulum usually precipitate an acute abdominal emergency. These complications include infection, perforation, hemorrhage, intestinal obstruction, volvulus, intussusception and neoplasm. It is estimated that pathologic changes occur in 15 to 25 per cent of cases of diverticula.

Infection. Acute inflammation of the diverticulum, similar to that of appendicitis, often is caused by the presence of an obstruction at the base of the organ, or by foreign bodies, with accumulation of secretions and the invasion of the wall by microorganisms. Clinically, such an inflammatory process usually is indistinguishable from symptoms arising from acute appendicitis. Vague, migratory, abdominal discomfort may result from mild or subacute inflammation and in such cases a diagnosis of Meckel's diverticulum may have to be considered. In our group of cases, however, such a patient was thought to have had recurrent attacks of appendicitis, until operation disclosed the true origin of the intra-abdominal disease. Inflammation, as the primary cause of symptoms, was noted in 9 patients, 3 of whom had peritonitis due to perforation (table 3).

TABLE 3

Patients with acute symptoms arising from Meckel's diverticulum

Diagnosis	Age	Sex	Gastric Mucosa		Preoperative Diagnosis and Remarks
			Yes	No	
Massive bleeding, 3 patients	22	M		x	Bleeding from Meckel's diverticula
	19	F	x		Bleeding, cause undetermined
	12	M	Not determined		Bleeding, cause undetermined
Obstruction, 2 patients	38	M		x	Regional ileitis, also present
	43	F		x	Band to umbilicus
Volvulus, 1 patient	24	F		x	Intermittent obstruction
Perforation, 3 patients	31	F	x		Developed peritonitis after operation on common bile duct, died, autopsy revealed a perforated Meckel's diverticulum
	27	M	x		
	18	F	x		
Inflammation, 6 patients	39	F	x		Acute appendicitis
	43	F		x	Cholecystitis; chronic Meckel's diverticulitis also found
	35	M		x	Recurrent appendicitis
	34	F		x	Regional ileitis; also present
	32	M	x		Atypical appendicitis
	38	F		x	Gangrenous Meckel's diverticulum found in hernial sac
Intussusception, 5 patients	9	F	x		Acute appendicitis
	3/12	M	Not determined		Intussusception—died second postoperative day of peritonitis
	49	M	x		Meckel's diverticulum with intussusception
	10 9/12	M	Not determined		Intussusception—died fourth postoperative day, blown stump, peritonitis
	44	M	Not determined		Intestinal obstruction

Deaths—3; total 20 patients.

Perforation. This complication may result from an advanced inflammatory state with gangrene, peptic ulceration, or perforation by a foreign body such as a fish bone. Ectopic gastric mucosa was found in both cases of perforation in our series, as it was in Berman's[2] cases (figs. 1 and 2). Since the location of the diverticulum is such that the *walling off process* is less easily accomplished than with appendicitis and the ileal content is more fluid and more copious, this peritoneal contamination may be more severe than the peritonitis resulting from a perforated gangrenous appendix. As noted above, we have had 3 patients with perforation. Frequently, acute appendicitis and perforated Meckel's diverticulum may seem to, or actually, coexist. Aalkjaer,[1] as quoted by Christensen, wrote, "In several of these cases no discrepancy has been found between the condition of the appendix and the peritonitis, and the surgeon has performed an appen-

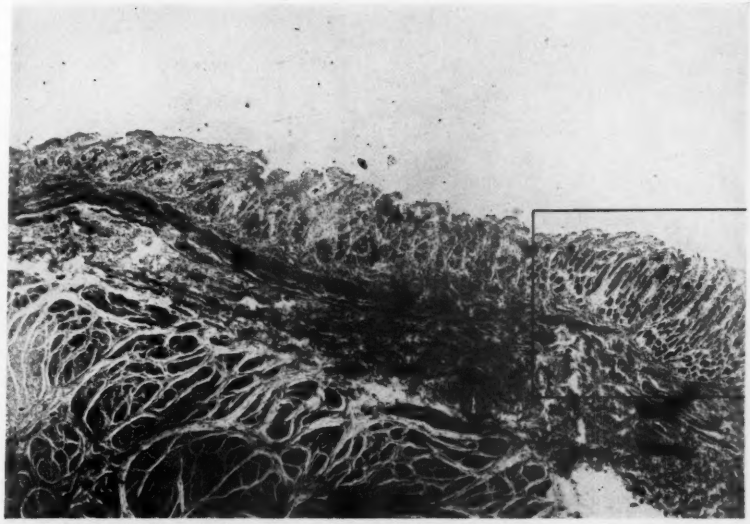

FIG. 1. Meckel's diverticulum from a 5 year old boy. Gastric mucosa is seen on the right, intestinal mucosa on the left (hematoxylin and eosin ×27.75).

dicectomy. Some days later necropsy has revealed the perforated Meckel's diverticulum." In 1 patient in our series a perforation of a Meckel's diverticulum developed after a secondary procedure on the common bile duct to arrest hemorrhage. The signs and symptoms of acute peritonitis developed three days after operation, and were thought to result from the intra-abdominal surgery, but at subsequent autopsy a diverticulum with a perforation was found. As noted above, this specimen contained ectopic gastric mucosa.

Hemorrhage. Heterotopic tissue such as gastric, duodenal, jejunal or even colonic mucosa is common in Meckel's diverticula but ectopic gastric mucosa occurs more frequently and is found in 10 to 20 per cent of the patients. Gastric mucosa alone, however, may lead to ulceration with consequent hemorrhage or perforation. Aberrant pancreatic tissue also is rarely found in Meckel's diverticula. The peptic activity of ectopic gastric mucosa within the diverticulum occasionally produces ulceration, and hemorrhage is a frequent result of such erosion. The ulceration usually occurs at the edge of the gastric glands or at the neck of the diverticulum. Gastric mucosa was found in 11 of 13 cases of massive hemorrhage in Berman's series but was seen only once in our 3 cases. In 1 of our cases previously reported,[11] the ulceration apparently took place in normal intestinal mucosa. Massive rectal bleeding should be suspected as coming from a Meckel's diverticulum when the usual studies of the stomach, duodenum, colon and rectum are repeatedly negative; if the bleeding persists, exploratory laparotomy is justified and should be carried out. This is particularly true in children under 10 years of age.

Intestinal Obstruction. This may be produced in a number of ways, but usually

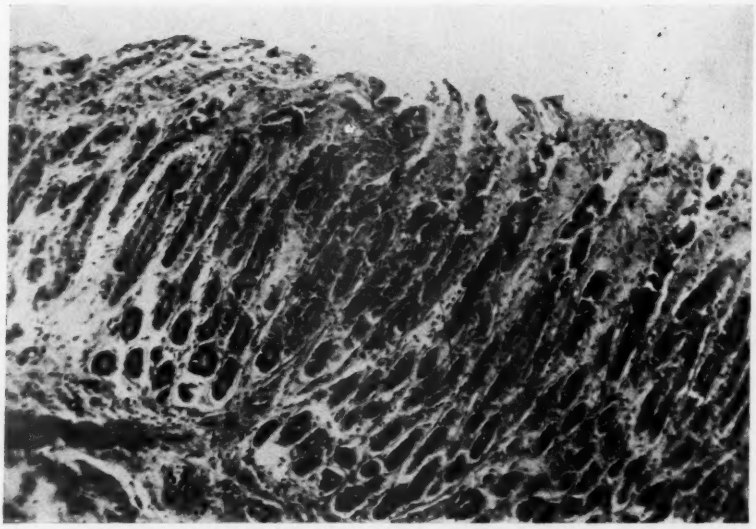

FIG. 2. Area of gastric mucosa (outlined in fig. 1) in Meckel's diverticulum under high power (hematoxylin and eosin ×87.5).

there must be a fairly mobile pouch or a band extending to neighboring ileum, umbilicus, or pelvic viscus. These may kink a portion of the bowel, form a loop around it, or create an aperture through which a segment of the intestine may prolapse and become strangulated. The presence of a strangulated Meckel's diverticulum in umbilical, femoral, and inguinal hernias has been noted by various authors as a cause of intestinal obstruction. One such case is included in our series.

Volvulus. The mechanism which produces volvulus is about the same as that mentioned under obstruction. A band to a nearby structure allows a loop of bowel to twist on itself and prolapse through the arch thus produced. The *closed loop* type of obstruction usually results with the characteristic violent intestinal colic, collapse, fever and leukocytosis. It is rare in most reported series. The 1 patient in our group of cases presented symptoms of intermittent obstructive attacks over a long period.

Intussusception. The diverticulum may provide the leading point of an intussusception, the clinical symptoms, signs and course being identical with intussusception due to other causes. Even though the intussuscepted bowel may be reduced by enemas, an exploratory laparotomy should be done and the diverticulum resected when the general condition of the patient permits, because of the possibility of recurrence of intussusception and of the development of other complications, principally, gangrene of the bowel. In 1 of our patients, the diverticulum causing the intussusception also was perforated. Figure 3 shows clearly the mechanism in 1 of our patients. The diverticulum had pouched inward and, acting like a polyp, had drawn the bowel 2 feet into the colon.

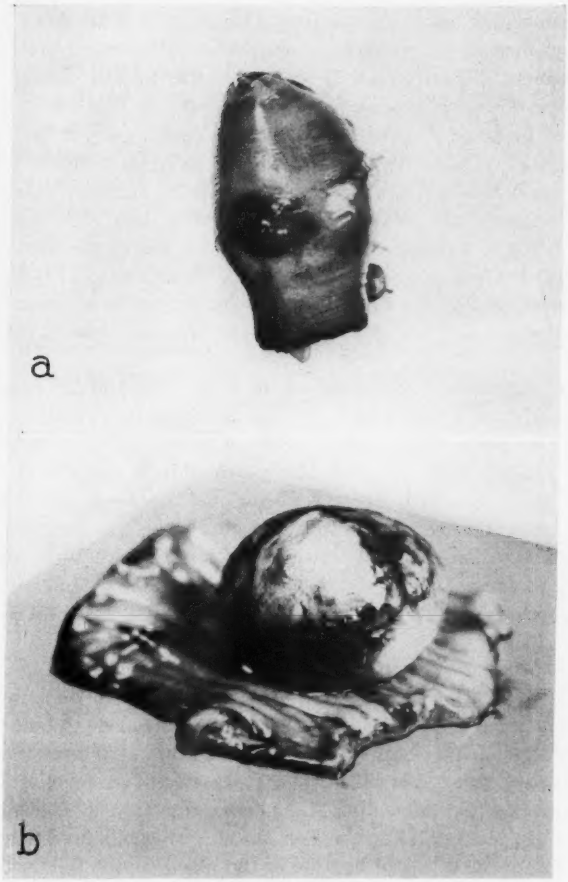

FIG. 3. *a*, Serosal surface of ileum revealing Meckel's diverticulum. *b*, Ileum opened to show invaginated Meckel's diverticulum which acted as lead point of intussusception.

Other Complications. Various types of neoplasms, notably carcinoids, have been described in Meckel's diverticulum, but others, such as carcinomas and benign and malignant tumors of connective tissue origin, also have been encountered.[13]

DIAGNOSIS

A preoperative diagnosis of a complication arising from the presence of a Meckel's diverticulum usually is presumptive, since most of the pathologic states are indistinguishable from similar complications arising in other organs in the vicinity, and there are no typical clinical findings helpful in differentiating basic causes of obstruction, intussusception or volvulus. As pointed out previously, hemorrhage from the diverticulum must be diagnosed by exclusion of the

possibility of bleeding from other sources. Occasionally a Meckel's diverticulum can be demonstrated by roentgen examination. Rousseau[14] stated that if a persistent segment of barium lies at an angle with the rest of the ileum and it does not exhibit the usual small bowel mucosal pattern, a Meckel's diverticulum may very well be present. Lewitan[10] reported a case in which the diverticulum was demonstrated on a roentgenogram, but the serial fluoroscopic films revealed that the location was not always constant. The changing position of the diverticulum explained the migratory nature of the pain, since in each case it corresponded to the location shown on the films. He emphasized the value of serial films to demonstrate the presence of a diverticulum. Bizarre symptoms such as those related above are a common finding by most observers, and the variation in size, location and position of the anomalies indicated helps to explain their basis.

Differential diagnosis is concerned, in the main, with the differential diagnosis of other abdominal emergencies. In the inflammatory states, appendicitis, perforated ulcer, acute cholecystitis, acute pancreatitis, mesenteric thrombosis, colitis, and related inflammations always should be considered. In bleeding states, hemophilia, Schönlein-Henoch disease, hemangiomas, small bowel tumors and polyps must be ruled out, as well as peptic ulcer, gastric and colon lesions. Mechanical aberrations, such as intussusception and volvulus, are not distinguishable as to cause, but very often present problems in differential diagnosis from inflammatory conditions arising from other pathologic states.

TREATMENT

When a complication resulting from a Meckel's diverticulum becomes an abdominal emergency, decision as to the surgical procedure to be employed must be based on a well defined surgical entity which is found at laparotomy. The only treatment is surgical excision. If inflammatory, exudative reaction is severe with marked induration and thickening of the ileum, resection of the ileal segment will be necessary. In most patients the excision of a Meckel's diverticulum is a very simple procedure consisting of ligation of the blood supply followed by clamping of the base of the diverticulum and closure with two layers of suture material, the second serosal layer consisting of interrupted sutures of fine black silk. The clamp is applied across the base of the diverticulum, not too close to the ileum so that inversion of the divided end will not produce narrowing of the ileal lumen. Simple inversion of Meckel's diverticulum with purse-string suture, as in inversion of an appendix stump, should not be done since this produces a polyplike tumor in the small bowel which may result in the development of intussusception. When a diverticulum is encountered incidentally, however, in the course of an unrelated abdominal procedure, all other contingencies must be evaluated before excision of the diverticulum is added to other intra-abdominal procedures. It the excision can be accomplished without the necessity for resection of the bowel to prevent narrowing of the intestinal segment, it always should be done to prevent possible future disturbance. If, however, excision is deemed unwise at the time of its discovery, the presence of Meckel's diverticulum should be

recorded in the operative note for reference in the event of later complication arising from it.

In view of the fact that Meckel's diverticula are common (1 to 2 per cent in autopsy material), it would seem wise always to search for diverticula during any abdominal surgical procedure. Excision of the diverticulum when found under such circumstances would not incur any greater risk than the incidental removal of the appendix during an abdominal operation. If Meckel's diverticula are searched for routinely, future serious intra-abdominal complications may be prevented, whereas simple excision would not increase the hazard of other intra-abdominal procedures.

SUMMARY

A report of 93 cases of Meckel's diverticulum has been presented. Fifty-one were found incidentally during other procedures, and 22 were found at autopsy for unrelated conditions. Twenty were excised as the primary source of intra-abdominal pathologic conditions.

Analysis of the remaining 20 cases occurring as surgical entities revealed that 8 specimens contained ectopic gastric mucosa. The importance of this finding has been presented and emphasized.

Meckel's diverticulum has been shown to be of importance in explaining vague abdominal symptoms, as the origin of some acute abdominal emergencies and as a source of massive bleeding from the gastrointestinal tract.

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VISCERAL INJURY DUE TO NONPENETRATING ABDOMINAL TRAUMA: A REPORT OF EIGHT CASES*

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Abdominal nonpenetrating trauma can be the precursor of a major catastrophe which may involve not only the recovery of the injured patient but also the reputation of the attending physician. Certain of these injuries, although apparently benign when first seen, are prone to grave sequelae. Minor symptoms or signs, initially, may be the only portent of serious complications from associated visceral trauma. More often, associated external injury (e.g., fracture and head injury) overshadow and distort the significance of blunt abdominal trauma. The attending physician, preoccupied with the care of the obvious injuries, may not recognize the insidious development of an acute abdominal condition which is mandatory of immediate treatment, usually surgical. In certain closed injuries the diagnostic acumen and judgment of the surgeon is severely taxed since the decision for or against exploration can be difficult. The wrong decision may be tragic.

This paper will include a discussion of some of the general problems relating to the importance of nonpenetrating abdominal trauma. Eight case reports then will be given from the files of the King County Hospital to demonstrate certain features of the syndrome of visceral injuries resulting from such trauma. These case reports are merely illustrative and do not represent all of such patients who were seen within the past few years in our hospital. For example, Knopp and Harkins[11] (1954) have reviewed 28 cases of traumatic rupture of the spleen seen at the King County Hospital from 1938 to 1951 (inclusive), 25 of which resulted from closed injuries.

GENERAL CONSIDERATIONS

Blunt trauma to the abdomen is seen commonly as the companion of the automobile accident whether it be driver, passenger or pedestrian who is injured. Collins[5] (1949) reported approximately 50 per cent of 225 grave injuries to abdominal viscera were caused by automobile accidents. Similarly, 61 per cent of the cases of traumatic pancreatitis, secondary to nonpenetrating trauma, reported by Berne and Walters[1] (1953), followed a car accident. By contrast, a patient in this latter series was treated for pancreatitis said to be secondary to the force of a stream of water from a garden hose applied to the abdomen.

Many common and unusual visceral injuries from various forms of acute blunt trauma to the abdomen have been reported. The experiences with rupture

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of the liver, spleen, and kidney are legion. In a study of 800 patients these so-called, fixed organs were involved in 70 per cent of the cases (Cottrell,[6] 1954). Stransky[19] (1954) described a case of retroperitoneal rupture of the duodenum due to nonpenetrating trauma to the abdomen; he reviewed the literature and adequately discussed the diagnostic problem and management. Similarly, Cottrell (1954) devoted considerable attention to this formidable complication. The problem of traumatic pancreatitis from blunt abdominal injury has been adequately surveyed (Naffziger and McCorkle,[15] 1943; Shallow and Wagner,[18] 1947; Mathewson and Halter,[14] 1952; and Berne and Walters, 1953). Similarly, gastrointestinal tract trauma has been extensively reviewed (Kirtland,[10] 1953; Cottrell, 1954; Johnston,[8] 1954, and Bradley,[2] 1954). Mason and associates[13] (1954) reported 2 cases of rupture of the extra-hepatic bile ducts from nonpenetrating trauma in infants, and Schaer and co-authors[17] (1955) described this injury in an adult. Additional cases were cited and both groups of authors discussed the management of this particular injury.

More unusual visceral injuries from blunt abdominal trauma are amply recorded. Hinckley and Albertson[7] (1954) described a successful resection of a gangrenous segment of ileum due to avulsion of the mesentery. Johnston (1954) presented 4 bizarre cases, 1 of which was a fatal small bowel obstruction from impingement of a loop of jejunum between the fragments of a fractured pelvis. A lacerated superior mesenteric artery was repaired by Ulvestad[20] (1954) with success. Lampert and associates[12] (1954) cited their experience with a 15 year old white boy who developed roentgenographic evidence of small bowel obstruction within 72 hours after a kick to the abdomen. At laparotomy the obstruction was found secondary to a large subserosal hemorrhage of small bowel.

These cases are cited to illustrate the occasional formidable complication of blunt trauma to the abdomen. In general, the greater the wounding force the greater the likelihood of internal visceral injury. Usually, the more severe internal injuries are associated with external and obvious trauma as fracture. The incidence of multiple visceral injury also is proportional to the severity of the trauma, although Mathewson and Halter (1952) cited 5 of 9 cases of traumatic pancreatitis in which the pancreas was the only organ involved. It also is recognized that the full hollow viscus, much as the urinary bladder, or the pregnant uterus, is prone to rupture from lesser degrees of trauma. However, many different types of severe visceral injuries have been reported from exceedingly minor abdominal trauma.

It is extremely important, therefore, that the attending physician be alert to the possibility of visceral trauma in this type of injury, which when mismanaged may bring grave sequelae. Unfortunately, as previously implied, the diagnosis is not always easy, nor are the indications for or against laparotomy well defined. Breidenbach[3] (1954) stated the problem in these words: "The difficulty with all blunt injuries to the abdomen is making the diagnosis, . . . mortality is still tremendous in blunt injuries to the abdomen." He then went on to state that most of the high death rate "is due to the fact that we procrastinate and are unable to make a diagnosis."

ESSENTIALS OF DIAGNOSIS AND TREATMENT

It is not our purpose to detail the methods of diagnosis and treatment of the various internal injuries one may encounter. Most of the authors already referred to have elaborated on these facets of management. It is essential, however, to spotlight the major clinical, laboratory, and roentgenologic studies, some mandatory, some essential, and all pertinent.

Two rules of thumb seem significant. First, we must think of the possibility of internal visceral trauma, especially where attention is primarily focused on concomitant obvious injuries. Second, when doubt exists as to diagnosis, repeated perhaps frequent examination of the patient clinically, with appropriate laboratory and roentgenologic study is rewarding.

Pertinent history as to the type of injury and the resultant symptoms is significant but in many cases unavailable. An evaluation of the total patient is important. Does his status suggest that something more than the obvious injury has occurred? The pulse rate, blood pressure, and temperature must be observed for clues. Are the symptoms or signs related to the abdomen? Even the minimal findings should be rechecked in a few hours to rule out progression. What are the physical findings relating to the abdomen; do they suggest peritonitis or intraperitoneal hemorrhage? Are the findings becoming more definite or worse? Has there been a progression in hypoactive bowel tones, minimal abdominal tenderness, or questionable muscle spasm, if present on the first examination?

The hematocrit and white blood cell counts should be noted and repeated after a short time interval. A urinary bladder catheter should be inserted unless one can be certain that no bladder or renal trauma has been sustained. Blood should be readily available for transfusion if necessary. Serum amylase and urine diastase studies should be made routinely where there is abdominal trauma. It is important to know during the acute phase of the treatment if these determinations are elevated. Later, they may be of great significance for medicolegal reasons should the problem of etiology of pancreatic cyst arise. Berne and Walters (1953) in their series of traumatic pancreatitis stated that pseudocyst of the pancreas developed in 3 of 14 patients who survived nonpenetrating trauma to the abdomen. In cases of liver damage the bilirubin level may be helpful in the evaluation of the extent of trauma. Abdominal needle puncture for intraperitoneal blood or bile may be indicated when there is doubt, but only a positive finding is significant.

A chest roentgenogram and flat, upright, and left lateral decubitis films of the abdomen, including the pelvis, are essential. Free intraperitoneal air may or may not be visualized in laceration of the bowel. Retroperitoneal rupture of the duodenum may show diffuse retroperitoneal gas or gas along the psoas shadow. A small quantity of lipiodol injected through an inlying gastric tube may visualize a duodenal laceration otherwise not detectable.

To summarize, some patients will demonstrate signs so obvious that few laboratory or roentgenologic studies are necessary and exploration is mandatory. Others will require the gamut of studies suggested above and perhaps their repetition. Initially certain patients will have mild symptoms and signs referred

to the abdomen, findings which will rapidly progress to acute *surgical* abdominal condition; others with similar mild findings will improve, and require only conservative therapy.

The treatment of traumatic pancreatitis is not unequivocally established. Therapy would be enhanced significantly if one could rely on present laboratory studies to indicate the extent of pancreatic injury (cf. case 1).

Shallow and Wagner (1947), Mathewson and Halter (1952), and Cottrell (1954) advised conservative treatment in instances of uncomplicated traumatic pancreatitis. Kirby and co-workers (1955) discussed the cases of secondary hemorrhage in hemorrhagic pancreatitis and after operative injury to the pancreas. One should be cognizant of such a complication when treating conservatively the pancreatitis from blunt abdominal injury.

When a reasonable doubt in diagnosis is present, it is better to explore those patients who have closed abdominal injury than to procrastinate. Clarke[4] (1954) stated that exploration must be made on mere suspicion alone. Particularly is this true in those cases in which traumatic pancreatitis is established by laboratory tests but a clinical suspicion of concomitant visceral injury is entertained. Rupture of a viscus is frequent enough in blunt abdominal trauma to warrant it as a tentative diagnosis and to operate even if there is only a well founded clinical suspicion. When one does explore an abdomen and finds evidence of pancreatic trauma, adequate drainage must be instituted. Sump drainage with collection and determination of enzymatic activity is preferred (Scott and Harkins,[16] 1951).

CASE HISTORIES

The hospital course of 2 patients who were injured from kicks to the abdomen is summarized. Both sustained severe internal trauma necessitating surgery. The third case is one of trauma to the abdomen from a hockey stick.

Case 1. W. E., a 26 year old white man, was kicked in the abdomen and beaten into unconsciousness. He was admitted to the King County Hospital in the morning of May 10, 1955, approximately 11 hours after the injury. He complained of abdominal pain and vomiting of gastric content. On examination he appeared moderately ill. His blood pressure ranged in the vicinity of 150/70, pulse 120 per minute, respirations 40 per minute, and temperature 99.8 F. The abdomen was slightly but diffusely tender with guarding but no true rigidity. There was rebound tenderness in the left upper and right lower abdominal quadrants. No masses were palpable. Bowel tones were absent. A stomach tube was inserted and suction applied. Preoperative laboratory data: Hematocrit, 45; white blood cell count, 21,200 per cu. mm., and serum amylase, normal. Chest roentgenogram and flat films of the abdomen were not remarkable. His blood pressure remained elevated and his pulse rapid. He received antibiotics, intraveous fluid, and blood. Without significant change of his condition in the next few hours, it was decided to operate (Dr. Lloyd M. Nyhus) for possible rupture of an internal viscus. Approximately 200 cc. of bloody fluid was aspirated from the abdominal cavity when opened. The body of the pancreas immediately to the left of the vertebral body was completely transected but the splenic vessels and spleen were intact. A moderate retroperitoneal hematoma was present. The body and tail of the pancreas distal to the point of transection was resected, the pancreatic duct ligated and the raw end of the pancreas covered with pancreatic capsule. The spleen also was removed. Sump drains were led from the area of injury through bilateral stab incisions. The day

after operation, the drainage from the abdomen measured only 128 cc. but contained over 1500 units of pancreatic amylase. This decreased rapidly and the tubes were removed five days after operation. His postoperative course was uncomplicated and he was discharged on May 21, 1955.

Comment. It is significant that a kick to the abdomen could transect the pancreas completely. Moreover, the serum amylase preoperatively was normal. Fortunately for this patient the aggregation of clinical clues was great enough to warrant exploration for possible viscus injury. Procrastination would have meant exploration eventually with a difficult and delayed convalescence, if not a fatal outcome. Because adequate drainage of the pancreas was established, the fluid, which, even though of low volume, had a high amylase content, could not pool in the area of injury. Recovery was thereby expedited and the danger of development of pancreatic cyst was minimized.

Case 2. This 32 year old white woman, an alcohol addict, was admitted to the King County Hospital for the second time on Aug. 13, 1954. Approximately 16 hours prior to admission and culminating an alcoholic spree of three weeks, she allegedly had been beaten and kicked in the ribs and abdomen. Initially, she had vomited bile-stained gastric juice but later vomited bloody fluid. In a previous hospital admission she was treated for a left lower lobe pneumonia and toxic psychosis. Examination showed an acutely ill, moderately well nourished, dehydrated female. Her skin was pale, cold and clammy. No blood pressure or radial pulse was obtainable but the apical rate was 120 per min. Respirations were 30 per min. and temperature, 101 F. She vomited blood during examination. There was extensive bruising of the left rib cage with obvious lower rib fracture. Breath sounds were normal. The abdomen exhibited clearly a marked periumbilical ecchymosis; it was rigid, tender, and without bowel tones. There were bilateral ecchymoses of the flanks. No masses were palpable. Hematocrit and urinalysis were normal. Portable roentgenograms demonstrated multiple fractured ribs on the left, lower left hemothorax, a left pneumothorax with approximately 10 per cent collapse of the lung, and free intraperitoneal gas. She was treated for shock and dehydration with intravenous fluids, plasma expander and whole blood.

A gastric tube with suction was inserted, and a Foley catheter was placed in the bladder. An intrapleural suction catheter was inserted in the left chest to expand the lung. She was started on intravenous terramycin, which was changed later to the intramuscular and oral forms when progress warranted it. The blood pressure rose to normal levels but her pulse remained rapid. Twelve hours after admission, under general anesthesia, her abdomen was explored (Dr. Ralph J. Schlosser). Approximately 2400 cc. of bloody fluid was aspirated from the abdominal cavity. Evidence of early peritonitis was present. A small laceration of the greater curvature of the stomach just proximal to the antrum was closed. The jejunum was edematous and heavy. Thirty-five cm. from the ligament of Treitz, the jejunum was lacerated involving 50 per cent of the circumference of the bowel. This was repaired satisfactorily. Bilateral sump-type drains were led out through flank stab incisions and the abdomen was closed with buried interrupted wire sutures reinforced with mattress sutures of wire. The postoperative course was complicated by atelectasis in the right lower lung field which cleared, and by a postalcoholic psychosis. All tubes were removed by the third postoperative day, there having been no drainage from the abdomen. She was discharged on August 23, 10 days after admission.

Comment. The indications for exploratory laparotomy were definite but were secondary to immediate therapy directed at shock and dehydration. When these abnormalities were corrected, the abdomen was explored. Procrastination could

have meant an extremely difficult convalescence after surgery or might have resulted in death. A secure wound closure was believed to be essential in this case of multiple visceral perforation. Again, it is significant that this extent of injury to the viscera was incurred from the trauma of a kick. Sump drains were employed but removed early when no drainage was recovered.

Case 3. D. D., an 11 year old white boy, was admitted to the King County Hospital Sept. 15, 1951, approximately 16 hours after being hit in the abdomen with a hockey stick. Initially there was only slight pain at the time of injury. Later, colic, vomiting, and an abdominal mass developed. He then was brought to the hospital and admitted immediately. Examination revealed an acutely ill child lying with legs drawn up. There was no evidence of shock, but the pulse was rapid (108 per min.). There was a well defined, tender mass 8 by 12 cm. in size of the right rectus region below the umbilicus. Peristalsis was normal. Rectal examination was negative. The first impression was a right rectus muscle hematoma but a contusion of the bowel could not be ruled out.

Laboratory data: Urinalysis, not remarkable; hemoglobin, 9.0 Gm.; white blood cells, 16,800 per cu. mm. Roentgenogram: No free air within the peritoneal cavity. Moderate amount of gas in midportion of the transverse colon.

The patient was transfused with 1 unit of blood and was operated upon (Dr. John Finley) two and a half hours after admission. A large hematoma was found overlying the cecum. When this was evacuated, a large laceration of the cecum was found. This extended down into the mesentery. The serosa and muscularis of the cecum were torn but at no point could a laceration into the bowel lumen be demonstrated. The tear in the bowel wall was closed without difficulty. The child did well postoperatively except for some paralytic ileus. He was discharged on the tenth day.

Comment. In this case, blunt injury to the lower abdomen at first seemed benign in character. Progression of symptoms, however, necessitated a hospital admission. Because an intraperitoneal viscus injury could not be ruled out, an abdominal exploration was done without delay. This showed an extensive injury to the cecum, a laceration which, interestingly enough, had not torn completely into the bowel lumen.

The above case histories were presented in some detail because of the occurrence of extensive visceral trauma from what one usually considers mild abdominal injury. Other interesting cases since 1951 have been seen at the King County Hospital, but with the exception of one, these have been associated with more extensive trauma.

Case 4. A 30 year old intoxicated Indian man from whom no history was obtained was explored for a possible abdominal viscus injury. A retroperitoneal rupture of the duodenum was found and repaired. The patient survived. Later, he stated a friend had told him he had been kicked in the abdomen.

Case 5. An 82 year old white man was injured by falling against a cement curb and immediately developed an acute condition of the abdomen. He was explored and a ruptured ileum was resected. The peritoneum had been generously soiled, in part by food particles. Later in his postoperative course a pus-filled left direct inguinal hernial sac was excised. This abscess had resulted from a piece of food contained in the direct sac. A modified repair of the defect was done. He was discharged 13 days after admission. He was last seen (Jan. 11, 1955) at the age of 84 years. During the intervening period he had been admitted three times and treated respectively for a cerebral vascular accident, a mild concussion, and a right lower lobe pneumonia.

Case 6. A 72 year old white woman was struck by an automobile. She sustained compound left tibial and fibular fractures and closed fractures of the right tibia and fibula, and pelvis. In addition an acute abdominal condition was present. An abdominal exploration was done and two small lacerations of the ileum were found. A segment of bowel containing both lacerations was resected. The fractures were treated conservatively. Six days after admission when her general status warranted it, she was transferred to private care for further care of the fractures.

Case 7. A 45 year old white woman was explored for possible viscus injury after sustaining blunt abdominal trauma when struck by a car. A rupture of the retroperitoneal duodenum was repaired. Some fat necrosis also was noted. Subsequently retroperitoneal drainage was necessary because of abscess. She was discharged on the twenty-third hospital day. This patient, in addition to the bowel injury, also had sustained a fracture of the lamina of the fifth lumbar vertebra. Postoperative serum amylase studies were normal.

Case 8. A 24 year old white man was severely injured in an automobile accident. He was admitted to the hospital in shock and with the findings of an acute abdominal condition. Despite adequate measures he failed to respond completely to antishock therapy. Abdominal aspiration was positive for intraperitoneal blood. Laparotomy disclosed a ruptured spleen and liver, complete and partial transection of the jejunum, laceration of the left leaf of the diaphragm and a laceration of the left renal vein. He remained in shock following operation and died on the day after admission.

Comment. These last 5 cases represent more severe injury except case 4 in which the history of trauma is unreliable. In none of these cases were preoperative serum amylase determinations made. Only in case 7 were determinaations made postoperatively. Amylase studies in this type of injury should routinely be obtained since definite assistance during the acute phase of injury may be realized. Later, they may be of value in medicolegal responsibility for pancreatic cyst, should one develop.

SUMMARY AND CONCLUSIONS

Nonpenetrating trauma to the abdomen must always be viewed with a high index of suspicion for possible internal visceral injury, regardless of the type or extent of abdominal injury. Seemingly innocuous blows to the abdomen may terminate in catastrophe if the development of an acute surgical abdominal condition from a lacerated viscus is unrecognized.

Diagnosis of these cases if difficult, but at the same time is urgent, since the indications for surgical exploration are dependent upon it. Frequent, total examination of the patient in whom doubt in diagnosis exists is the rational and rewarding method of managing these patients.

Two case histories illustrative of severe visceral injury from the trauma of kicks of the abdomen are cited. A third case is detailed in which severe visceral injury stemmed from a minor hockey stick blow to the abdomen. Five additional interesting cases are briefly presented. Other visceral injuries reported in the literature, many of which are unusual or resulted from mild blunt abdominal trauma, are mentioned.

It is hoped that increased alertness by attending physicians and appropriate, early surgical intervention, when indicated, will lower the high mortality rate of the complicated closed abdominal injury.

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THE JAUNDICED PATIENT

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The patient with jaundice frequently offers a challenge in diagnosis, not only to the internist and surgeon who are responsible for the method of therapy to be advised, but also to the clinical pathologist, who frequently casts the deciding vote as to whether or not it is a medical or a surgical problem.

Jaundice may be divided into many categories, but for practical purposes, the all important decision to make is whether it arises from a condition which should be treated conservatively or whether it results from a condition which may be relieved by surgical intervention. This is of great importance to determine, if possible, because exploratory celiotomy in the presence of hepatitis may result in a fatality and an unrelieved extrahepatic biliary obstruction will ultimately achieve a similar end result.

In approaching this problem, it is well to bear in mind that the individual's outcome with jaundice is rarely jeopardized by a period of observation, so it is much wiser, when in doubt, to ponder over the accumulated and accumulating data than it is to hastily explore the abdomen to find that surgery has nothing to offer.

From an anatomic and physiologic point of view, jaundice is most simply classified as being hemolytic or prehepatic, hepatic, hepatocellular or intrahepatic and obstructive or posthepatic. Hemolytic jaundice is relatively rare and usually can be recognized by consideration of the history, physical findings and red cell fragility studies. Being of relative unimportance both in frequency and as a diagnostic problem in comparison with hepatocellular and obstructive jaundice, it will occupy a relatively unimportant role in this discussion.

In order to approach the problem of jaundice intelligently, it is necessary to have at least a rudimentary grasp of the normal disposition of bile pigment and some understanding of what takes place when this mechanism is interfered with, resulting in jaundice.

Bile pigment arises from disintegration of red blood corpuscles and is brought about by the activity of the reticulo-endothelial system which, for the most part, lies outside the liver. The disintegration of the red blood corpuscles liberates the hemoglobin and this by various steps not pertinent to this discussion ultimately becomes bilirubin. The circulating bilirubin consists of two forms, the so-called direct and indirect; this distinction can be made by the van den Bergh reaction. In the direct type the diazo-reagent of Ehrlich (diazobenzosulfochloride) in aqueous solution gives a color reaction which occurs promptly. Indirectly, acting bilirubin produces a color reaction with the reagent only upon addition of alcohol, and the color is slower in reaching its maximum development. This chemical difference is thought to depend on the firmness of the association of the pigment with a serum protein fraction, the indirect type of bilirubin being firmly

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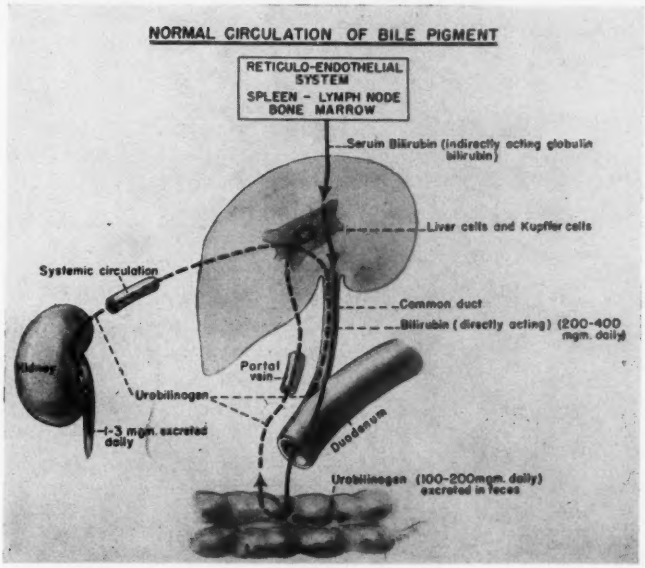

FIG. 1

bound to the protein while the direct is loosely attached. This explanation is not universally accepted. It is the belief of some at least that the direct type of bilirubin represents bilirubin which has passed through the hepatic cells, the indirect not having undergone this pilgrimage. One gram of hemoglobin, when broken down, produces 40 mg. of bilirubin and in 24 hours this results in 200 to 400 mg. of bilirubin being excreted by the liver. Figure 1 represents the normal process of circulation and excretion of bile pigment. Bilirubin from the liver passes down the common duct into the duodenum. In the intentine, by action of bacteria, bilirubin is converted into urobilinogen. The quantity of urobilinogen in a 24 hour stool in a normal person varies from 100 to 250 mg. Some of the urobilinogen is returned to the liver by the portal vein and is re-excreted as bilirubin or some other pigment complex by way of the common duct into the intestine. The normal liver does this so efficiently that less than 3 mg. is excreted by the kidneys in 24 hours.

HEMOLYTIC JAUNDICE

In prehepatic or hemolytic jaundice, these relationships are disturbed and figure 2 depicts the cycle of events that takes place under these circumstances. The breaking down of the red blood cells is greatly accelerated and results in an increase of bilirubin from a normal of around 200 to 400 mg. to as much as 1500 mg. or more in 24 hours. This results in a corresponding increase of urobilinogen being formed in the intestinal canal, the quantity varying with the severity of the disease. The increased quantity of urobilinogen in the intestine results in

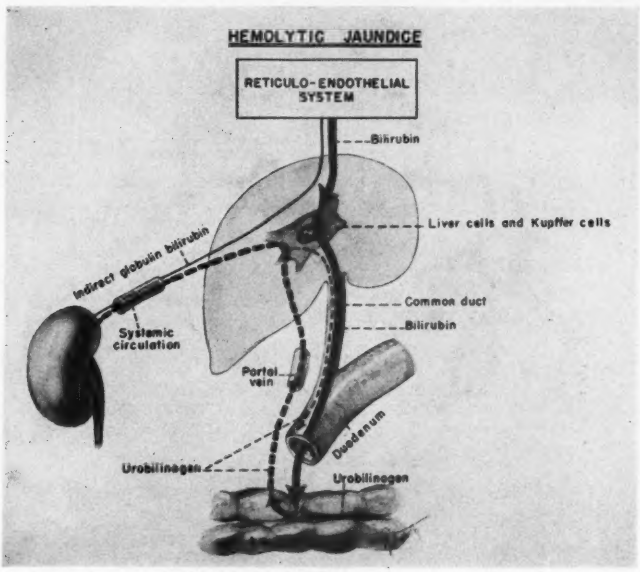

FIG. 2

increased absorption and return of urobilinogen to the liver via the portal vein. If the liver, under these circumstances, is unable to excrete the increased load of urobilinogen, urobilinogen concentration occurs in the systemic circulation and excretion by the kidneys is increased. An increased quantity of urobilinogen excreted in the feces and simultaneously in the urine is indicative of a hemolytic process.

Another characteristic of this type of jaundice is the presence of hyperbilirubinemia and the absence of bilirubinuria. This occurs because the liver is overworked and unable to convert bilirubin globulin, the indirect type, into the direct type which it can excrete. Therefore, the hyperbilirubinemia is due to the delayed or indirect reacting type of bilirubin and, because of the character of the pigment, it is not excreted in the urine; hence the term acholuric jaundice. This accounts for the characteristic indirect reaction of the van den Bergh test in hemolytic icterus. If marked bilirubinuria does occur under these circumstances, it indicates a hepatic or posthepatic factor. Since patients with hemolytic icterus are prone to produce gallstones, it is always possible that common duct obstruction may occur to confuse the clinical as well as the laboratory aspects of this condition.

HEPATIC OR HEPATOCELLULAR JAUNDICE

In this type of jaundice the usual quantity of bilirubin is delivered to the liver but because of the liver cell damage and the obstruction of the bile canaliculi from edema and necrosis, bilirubin is not completely excreted in the extra-

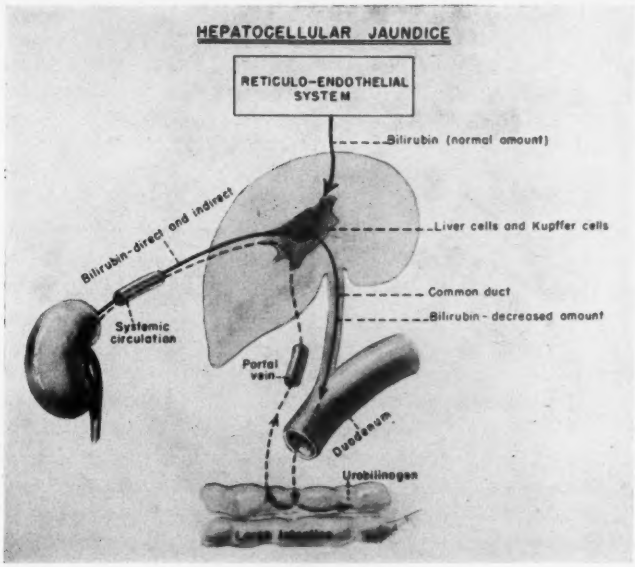

FIG. 3

hepatic bile ducts. Because of this obstruction and damage to liver cells, an increase in both the direct and indirect reacting type of bilirubin occurs. The intestine receives a decreased amount of bilirubin; therefore, a decreased amount of urobilinogen is formed. Bilirubin that has passed through the liver cells is dammed back by the obstructed intrahepatic bile ducts and therefore passes into the systemic circulation and appears in the urine as bilirubin. Urobilinogen that is formed in the intestine and reabsorbed into the portal vein cannot be excreted by the damaged liver and therefore passes into the systemic circulation and is excreted by the kidneys. If the obstructive process in the liver becomes complete enough, outflow of bile through the common duct may cease, the stools becoming acholic. No urobilinogen can be formed and the picture so far as bile pigment study is concerned resembles that met with in posthepatic jaundice. This situation usually is transient, the typical picture of hepatocellular jaundice returning, bilirubin appearing in the urine and urobilinogen in amounts above normal. Figure 3 represents the situation usually encountered in hepatocellular jaundice.

POSTHEPATIC JAUNDICE

In this type of jaundice the picture so far as bile pigment circulation is concerned depends upon the completeness of obstruction. If obstruction is complete and continuous, as frequently occurs in malignancy, no bilirubin passes into the intestine; hence no urobilinogen is formed and the stool becomes acholic (clay-colored). The bile regurgitates back from the extrahepatic ducts into

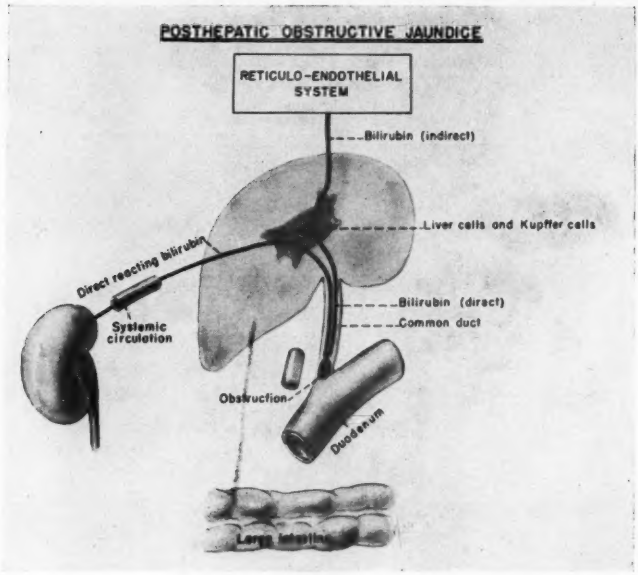

FIG. 4

the blood stream. Having passed through the liver cells, most of the bilirubin is of the directly acting type. The urine contains a large quantity of bilirubin but no urobilinogen. Figure 4 represents this type of jaundice diagrammatically.

In the early stages of extrahepatic biliary obstruction, the bile pigment studies may be diagnostic, but with long continued obstruction and damming back of bile, liver cell destruction occurs and the picture may be further complicated by the onset of infection and cholangitis. Under such circumstances, the study of the behavior of bile pigment may simulate the situation found in hepatocellular jaundice and be of no aid, even quite misleading in attempting to determine whether or not the case falls into the category of medical or surgical jaundice.

Keeping in mind this brief preliminary presentation of the generally accepted explanation of the formation and circulation of bile pigment in the various types of jaundice, one is in a better position to understand the three methods of approach that are at the present time available to us for differential diagnosis when confronted by a patient with jaundice. These are: (1) clinical (history and physical examination), (2) laboratory studies, and (3) microscopic study of biopsy specimens of the liver.

CLINICAL ASPECTS OF JAUNDICE

As previously mentioned, the most important thing to determine about the jaundiced patient is whether or not his jaundice falls into the category that will respond favorably to surgical intervention. Therefore, from a clinical point

of view, we may speak of medical and surgical jaundice. The history and physical examination is of extreme importance and may, and in fact usually does, contribute more to the differential diagnosis than does laboratory investigation. By this means alone it has been estimated that a correct diagnosis should be arrived at in 60 to 80 per cent of cases of jaundice.

MEDICAL JAUNDICE

The most common cause of medical jaundice is hepatitis and the most common etiologic factor causing hepatitis is a virus. These virus infections are conveniently divided into infectious hepatitis and homologous serum hepatitis. Infectious hepatitis is the terminology commonly used when speaking of the sporadic case while infectious epidemic hepatitis applies to the same disease when occurring in outbreaks.

While infectious hepatitis is not a new disease, it has in recent years, particularly during and since World War II, given rise to increased interest and investigation. It has been said that Hippocrates knew and wrote about contagious jaundice and that Pope Zacharias and Saint Boniface in the eighth century A.D. described an epidemic of jaundice. It has been recently determined by various investigators that infectious hepatitis is caused by a specific group of viruses. This investigation has for the most part been made on human volunteers, since no susceptible laboratory animal has been found. The virus is present in the stools and serum during the preicteric and early icteric stages of this disease. The disease is world wide, all ages being susceptible but is most commonly encountered between the ages of 6 and 40 years. Infectious hepatitis and serum hepatitis seem to be caused by similar but antigenically different viruses. It has been observed that volunteers infected with the infectious hepatitis virus had hepatitis following the first dose of virus but not following a second exposure. The same volunteers, if given the virus of serum hepatitis, developed jaundice with hepatitis but had no reaction following a second exposure to this virus.

Homologous serum hepatitis includes cases of viral hepatitis which have been accidentally or artificially produced as a result of introduction of blood or blood derivatives from an individual infected with this type of virus. Such infections at times occur in the laboratory following improper sterilization of needles that have been used on patients with virus diseases. Clinically this type of hepatitis is very similar to that produced by the virus of infectious hepatitis and the pathologic lesions produced are indistinguishable. The incubation period of infectious hepatitis is 20 to 40 while that of serum hepatitis is from 60 to 120 days. Hepatitis following vaccination against yellow fever, which we observed locally during World War II, was shown to be due to virus contamination of pooled human serum used in preparing the vaccine. Hepatitis and jaundice ceased following such inoculations when the use of human serum in its manufacture was discontinued. It is important, therefore, to inquire of the patient with jaundice if he has been the recipient within the past few weeks or months of blood or blood derivatives such as plasma or if he has been associated recently with anyone who was jaundiced. Also the question of drugs should be in-

quired into because some of these such as carbon tetrachloride, methyl testosterone, thorazine, arsenic, gold salts and others do at times produce hepatitis and jaundice.

One of the most important symptoms of virus hepatitis is weakness which usually is evident before the onset of jaundice. Malaise, anorexia and gastrointestinal upset are frequent symptoms. Dull aching pain in the region of the enlarged, tender liver frequently is complained of and is accentuated by percussion. Most patients have fever for the first few days and many have acholic stools during this time. Individuals suffering from jaundice of virus origin fall into a younger age group than those jaundiced secondary to extrahepatic obstruction. There are many causes and many aspects of medical jaundice which time and space do not permit discussing in a presentation such as this.

SURGICAL JAUNDICE

Surgical jaundice results from a number of causes that interfere with the flow of bile from the extrahepatic biliary ducts into the duodenum. The most common causes are gallstones, and carcinoma involving the head of the pancreas, the ampulla of Vater, the gallbladder or even the ducts themselves. Strictures of the ducts resulting from surgical trauma, secondary to stones and infection, and extrinsic pressure from various sources more rarely give rise to jaundice. Surgical jaundice frequently is not associated with pain and this suggests that it is due to malignancy, and jaundice associated with pain suggests gallstones. The patient known to have gallstones who develops sudden severe pain in the right upper quadrant, followed in 48 to 72 hours by jaundice, in all probability has a gallstone impacted in the terminal common duct. The patient of middle age or later who may not have been feeling up to par, perhaps has lost weight, who develops jaundice without pain in all probability has a malignant lesion obstructing the common duct. This is particularly liable to be the case if the gallbladder is palpable (Courvoisier's law). The presence of jaundice in a patient with stones in the gallbladder is not assurance that stones are present in the common duct. Jaundice in such patients frequently results from cholecystitis giving rise to cholangitis. It is, however, an indication to explore the common duct at the time of cholecystectomy. The absence of jaundice also is no assurance against the presence of common duct stones because in approximately 50 per cent of patients with common duct stones no history of jaundice is obtained.

While the history and physical examination may be of great help in determining whether or not a patient should be subjected to surgery, it frequently happens that the result of such an investigation is equivocal. Particularly under such circumstances, the internist and the surgeon then turn to the laboratory with the hope that additional information can be obtained that will help in this dilemma. After a number of experiences with such situations as described, the clinician may lose his enthusiasm for the assistance he may hope to get from this source. This may be due to the fact that the laboratory investigator is called too late in the course of the disease. As pointed out pre-

viously, simple obstruction of the common duct may soon be complicated by hepatocellular damage, the result of back pressure and infection. Hepatocellular jaundice usually is accompanied by a variable degree of abstruction of the intrahepatic bile ducts from edema. Hence, diagrams that clearly depict the behavior of bile pigment under various pathologic conditions affecting the liver, such as have been shown, frequently leave much to be desired when attempting to correlate them with the results appearing in the test tube.

We should, at least, be familiar with the usual procedures that are invoked and the tests that are most likely to throw light on the cause of jaundice, particularly if the cause seems doubtful clinically. The five most useful tests in estimating liver function and distinguishing between the various types of jaundice we have found to be are: (1) serum bilirubin (van den Bergh test); (2) determination of bromsulphalein retention; (3) qualitative and quantitative urinary urobilinogen; (4) flocculation tests; and (5) alkaline phosphatase.

SERUM BILIRUBIN

As previously mentioned, there are two forms of bilirubin in the blood stream depending upon the response produced when exposed to Ehrlich's diazoreagent. Directly acting bilirubin gives a quick color reaction and theoretically at least represents bilirubin that has passed through the liver cells. Indirect or delayed acting bilirubin only reacts with the diazo-reagent after the protein (globulin) with which the bilirubin is combined is treated with alcohol. This bilirubin has supposedly not passed through the liver cells. The normal values of serum bilirubin are 0.2 to 0.8 mg. per cent of which 0.2 to 0.3 mg. per cent is directly acting.

The van den Bergh test has its most specific application in determining the presence of hemolytic jaundice. In this condition, the total bilirubin is high and most of it is indirectly acting. In some instances, although apparently very rarely, the van den Bergh test is said to be of help in differentiating hepatocellular jaundice from posthepatic jaundice. Classically one should obtain an increase in the delayed (indirect) reacting bilirubin in hepatitis and an increase in the prompt directly reacting bilirubin in obstructive jaundice. From a practical point of view, this test is of little if any value in differentiating medical from surgical jaundice in most instances because, by the time the assistance of the laboratory is called upon, the increased bilirubin is almost always of the directly acting type. However, in any patient with jaundice this test tells how severe the jaundice is and serial determinations reveal whether the jaundice is increasing or decreasing.

It behooves the clinician to be alert to the possibility of aid from the laboratory if such tests are to be made before extrahepatic biliary obstruction gives rise to liver damage and intrahepatic obstruction which confuses the picture. The determination of total bilirubin in patients without clinical jaundice may be of importance in detecting early liver cell damage. Readings above 1 mg. per cent point to impairment of liver function. An elevation of the directly acting fraction with a normal total bilirubin has been reported in early cases of infectious hepatitis.

BROMSULPHALEIN

The bromsulphalein test generally is recognized as one of the most satisfactory and sensitive tests of liver function presently available. It is useful only in the absence of jaundice. The dye rarely produces toxic symptoms. The test is simple and, due to the rapidity with which it can be performed, it is useful in helping to differentiate hemorrhage resulting from esophageal varices accompanying cirrhosis of the liver from other types of upper gastrointestinal bleeding. In this clinic 5 mg. of the dye per kilogram of body weight are given intravenously and 45 minutes later the amount of retention is determined. The dye remains in the blood stream until it is removed in a progressive manner (apparently by the parenchymal cells of the liver) and excreted into bile and eliminated in the feces. Retention values greater than 5 per cent indicate impaired liver function and the degree of retention is roughly proportional to the extent of liver dysfunction.

UROBILINOGEN

Urobilinogen is formed in the intestine by the action of bacteria upon bilirubin. Normally the greater part of the urobilinogen thus formed is excreted in the stool (as urobilin), the remainder being reabsorbed by way of the portal vein and excreted by the liver which does it so efficiently that only small quantities appear in the urine. If the liver becomes damaged, it is unable to dispose of this reabsorbed urobilinogen and therefore this function is taken over by the kidneys, the urinary urobilinogen rising accordingly (fig. 3). A 24 hour urinary urobilinogen value of over 3 to 4 mg. can be considered abnormal and is indicative of hepatic dysfunction if hemolysis is ruled out. In extrahepatic obstruction urobilinogen is low or absent depending upon the degree of obstruction since the usual amount of bilirubin fails to reach the intestine. Our laboratory has been in the habit of doing a qualitative test on the urine, reporting the results in units depending upon the intensity of the color with the aldehyde reagent. Quantitative determinations on a 24 hour urine specimen also are done although precautions must be taken to prevent oxidation of urobilinogen to urobilin, and the collecting of a 24 hour specimen is a chore for the patient. Failure by the patient to be accurate may lead to false conclusions. Fecal urobilinogen also is done in some laboratories and may be the best procedure of all in determining how much bile is reaching the intestine.

FLOCCULATION TESTS

The principal flocculation tests used are the thymol turbidity and flocculation, the cephalin flocculation and the Kunkel. These tests are positive in more than 90 per cent of patients with parenchymal liver disease (hepatitis), and since their results have much the same significance, the thymol test is preferable since it can be completed in 30 minutes. It may not be quite as sensitive as the cephalin flocculation test in beginning hepatitis and takes longer to return to normal when the hepatitis has subsided. In some laboratories, the Kunkel zinc sulfate flocculation test is preferred.

ALKALINE PHOSPHATASE

It has been found that in approximately 90 per cent of patients with obstruction of the common duct the alkaline phosphatase in the serum will rise above 10 Bodansky units. In other types of jaundice it is more likely to be normal or only slightly elevated. The mechanism of this phenomenon has not yet been adequately explained.

To sum up these tests briefly: In the presence of jaundice where the problem exists as to its being medical or surgical, we have found that the serum bilirubin, urinary urobilinogen, the thymol flocculation test and the alkaline phosphatase determinations are the most useful. In the absence of clinical jaundice, the bromsulphalein test and the serum bilirubin, with determination of both direct and indirect fractions probably are of most significance. The determination of the prothrombin activity of the serum and its failure to rise in reponse to vitamin K administration is important evidence of hepatocellular damage but it does not indicate the type of liver damage if a low level exists.

LIVER BIOPSY

No doubt one of the greatest advances in the study of liver diseases, particularly as related to the differential diagnosis of jaundice, has been the development and perfection of needle biopsies. Until a few decades ago, most of the morphologic data relative to the liver had been obtained from autopsy studies and therefore was of no direct clinical importance in the study of a given patient. While surgical exposure of the liver presents a much more satisfactory means of investigation of this organ and those surrounding it and allows adequate removal of liver tissue in the most desirable area, it is a procedure not well tolerated by most individuals with liver disease. For this reason, the more recent use of various types of needles which are so constructed that they permit removal of fairly adequate specimens of liver tissue with relatively little danger to the patient has been receiving the increasing approval of all concerned, and no doubt it is a method of obtaining information deserving of wider use. It also has been suggested that the study of the material thus obtained differs from that of the autopsy room and may introduce problems in orientation and interpretation which require experience. It is immediately evident that specimens of liver tissue obtained in diffuse processes, such as cirrhosis and various types of hepatitis, are more likely to be diagnostic than in localized lesions such as neoplasms, granulomas because of the difficulty in securing the diseased tissue. As with liver function studies, biopsy studies, particularly as related to hepatitis, are more likely to be diagnostic in the early stages of the disease.

Needle biopsy of the liver is of particular value when the diagnosis of the cause of jaundice is in doubt after the clinical, roentgen and laboratory examinations have been completed. This method of investigation is especially useful in differentiating between hepatocellular and obstructive jaundice. It does not enable the pathologist to differentiate the cause of the obstruction, but it is of great value in helping to distinguish hepatocellular jaundice due to cirrhosis from other types of hepatitis. The accompanying photomicrographs illustrate a

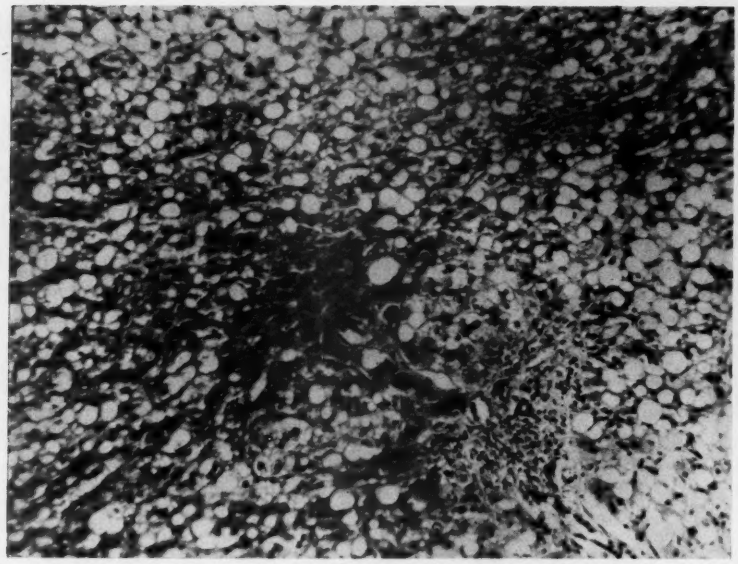

FIG. 5. Fatty metamorphosis of liver. Most of the liver cells contain fat. The architecture still is fairly well preserved although there is beginning fibrosis in the portal areas. The patient was a 43 year old man, an alcoholic addict, with no symptoms or findings except a greatly enlarged liver. Tests showed only minimal impairment of liver function and there was no jaundice. The liver has decreased greatly in size with medical treatment and abstinence from alcohol. Needle biopsy. Hematoxylin and eosin. ×120.

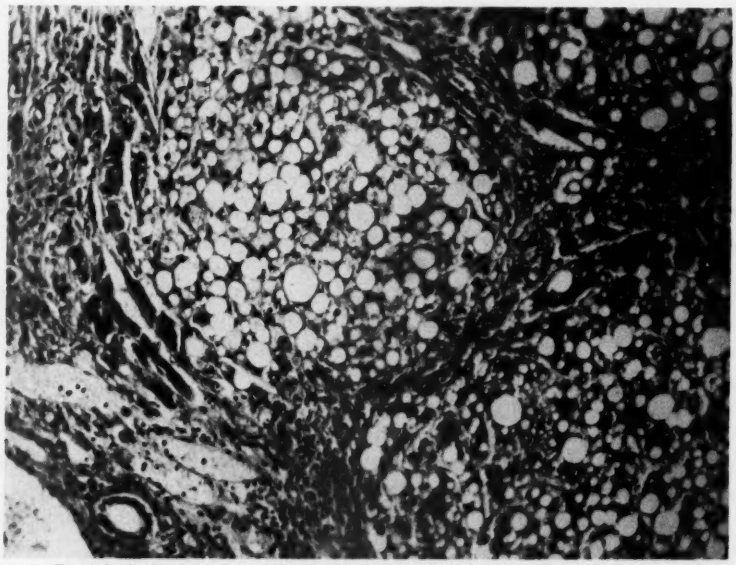

FIG. 6. Portal cirrhosis, early, in an alcoholic addict. The architecture is completely destroyed and fat is present within the nodules of regenerated liver cells. This man, aged 48, was known to have a large liver five years previously. Death occurred from ruptured esophageal varices. The liver weighed 2,100 grams. Hematoxylin and eosin. ×120.

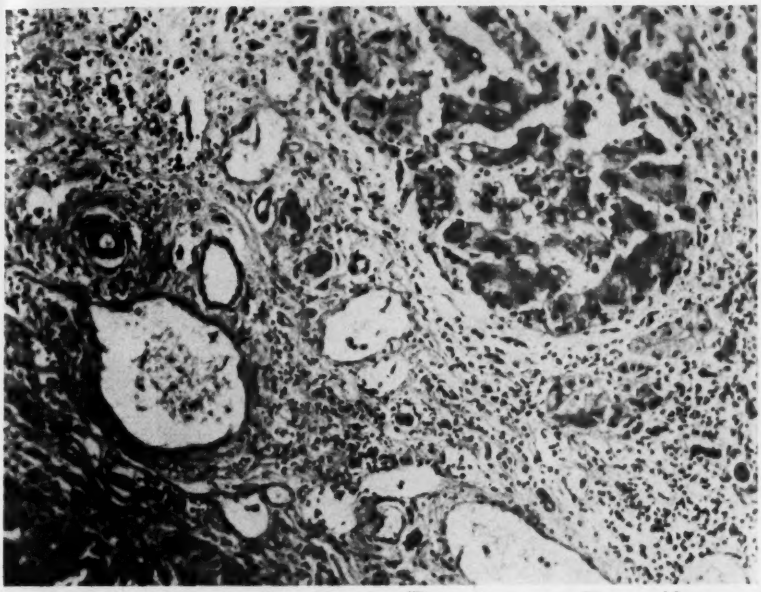

FIG. 7. Portal cirrhosis, late atrophic stage. The patient was a 53 year old man and a nonalcoholic. The liver architecture is completely disrupted with pronounced fibrosis, dilatation of the portal vein radicles, and isolated nodules of regenerated liver cells. The liver weighed 750 grams. Hematoxylin and eosin. ×120.

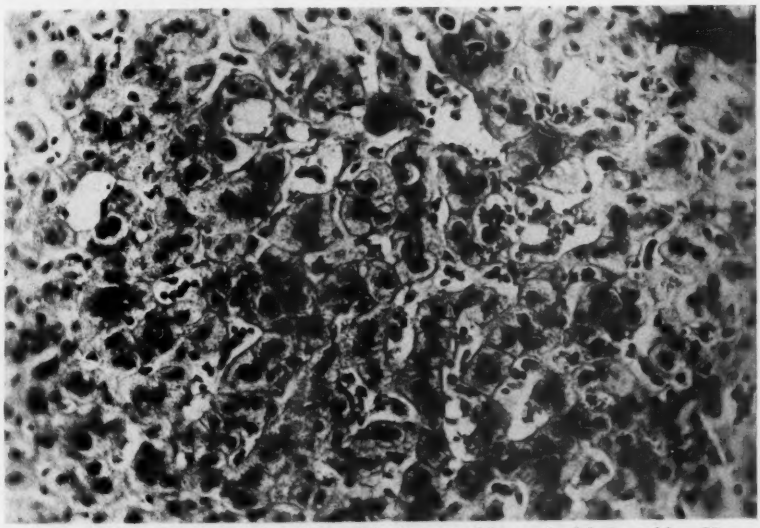

FIG. 8. Methyl testosterone jaundice. This 48 year old man had been taking 20 mg. of methyl testosterone sublingually daily for two months and developed severe jaundice with a bilirubin level of 8.4 mg. per cent. The question arose as to whether or not this was a case of medical or surgical jaundice. A needle biopsy was done. Examination of the needle biopsy by Dr. H. Civin showed dilatation of bile canaliculi with biliary retention and bile thrombi, but no disruption of architecture and little evidence morphologically of liver cell damage. It was thought to be compatible with cholangiolitic obstruction rather than extrahepatic obstruction. The patient made an uneventful recovery. Hematoxylin and eosin. ×265.

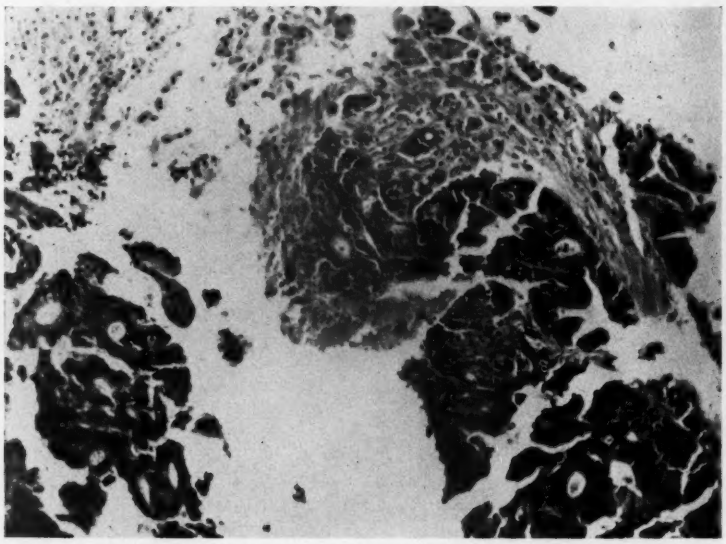

FIG. 9. Metastatic adenocarcinoma of liver. Liver biopsy. The malignant cells are shown below, liver cells above. Hematoxylin and eosin. ×120.

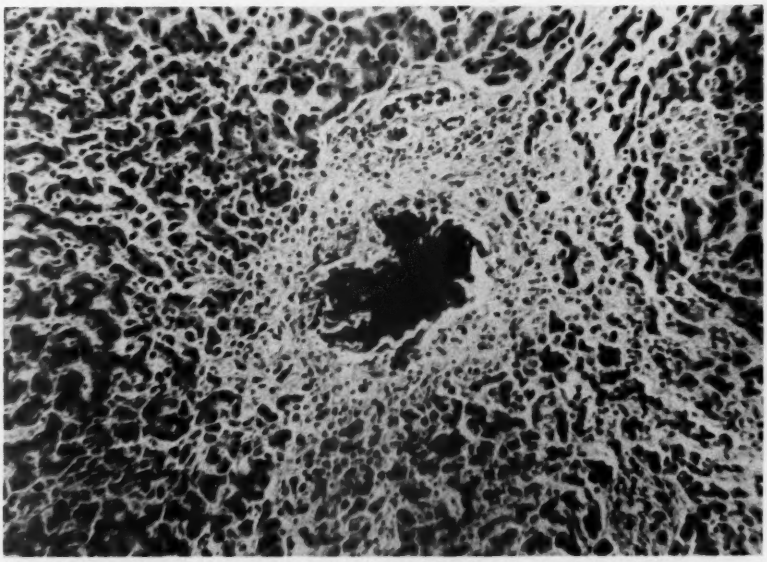

FIG. 10. Jaundice due to extrahepatic obstruction from metastatic adenocarcinoma of the stomach in a 70 year old man. Note dilatation of bile duct in portal area by inspissated bile with disruption of the bile duct epithelium. The liver cells show some damage toward the periphery of the lobule, but in the midzone area they are well preserved and appear normal. Hematoxylin and eosin. ×120.

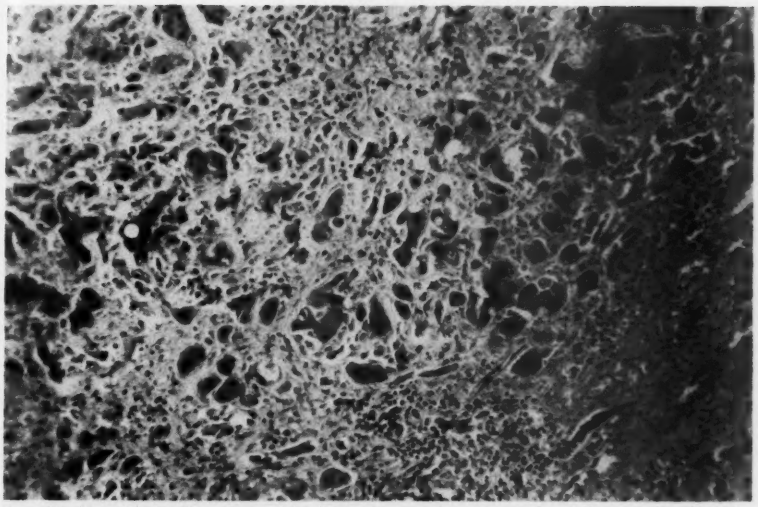

FIG. 11. Fatal infectious hepatitis. Note tremendous necrosis of liver cells with fibrous replacement, bile duct proliferation and lymphocytic infiltration. In former years this process was called acute yellow atrophy, but it now is regarded as fulminating infectious hepatitis. Hematoxylin and eosin. ×120.

few of the conditions in which needle biopsy of the liver may be helpful in establishing the diagnosis (figs. 5 to 11).

SUMMARY

It is evident from this presentation of the subject of the jaundiced patient that there are yet many unsolved diagnostic problems that frequently are encountered that do not lend themselves to ready solution. The liver is too complex an organ physiologically, its powers of regeneration and recuperation too great, and its functions (which are many) too frequently altered by a multitude of diseases to permit of easy mastering of the diagnostic problems in which it frequently is involved. A careful inquiry into the patient's history followed by a careful physical examination will in most cases permit a fairly accurate decision as to whether or not the patient is suffering from jaundice that may lend itself to surgical correction. These are the most important steps in arriving at such a decision. It is always advisable to remember that if the clinical pathologist is to approach these problems intelligently he should be acquainted with the clinical findings and his services should be enlisted at the earliest possible moment so that he may interpret his findings in the light of the early changes and not when the disease has resulted in the complicating factors previously discussed. As Fennel has so succinctly put it: "To the clinician who is too busy with overwork or just too lazy to get an accurate history or an estimate of the time or the stage of the disease which the patient presents, the laboratory findings will be about as useful as hip pockets are to hogs."

In our present state of knowledge, we must accept the fact that certain patients with jaundice will not fit into either surgical or medical group even after all methods of investigation now at our command have been exhausted. Under these circumstances, it will be advisable to subject some of these patients to exploratory laparotomy, fully realizing the dangers this may entail.

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CHRONIC CHOLECYSTITIS

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In our contact with patients having chronic cholecystitis, we have been impressed with the short duration of the individual attacks of pain. Although the surgical textbooks vary in their statements regarding the duration of pain in chronic cholecystitis, most of them either say nothing about the duration of attacks, or remark that each episode lasts from a few hours to a few days. Most clinicians apparently think of attacks of gallbladder *colic* as lasting from 12 to 48 hours. To investigate this and other phases of chronic cholecystitis, a study was made of 211 consecutive cholecystectomies done, either alone or in combination with choledochostomy, at the Illinois Research and Educational Hospitals. After reviewing the data in the records and questionnaires, we discovered that in a surprisingly high percentage of patients, 41.6 per cent, the attacks of pain lasted less than one hour. This has significance from two or three standpoints. In the first place, when the attack is of short duration there will be a tendency to ascribe it to some insignificant cause, such as dietary indiscretion; accordingly, on such occasions, procurement of a correct diagnosis is delayed. We likewise should appreciate that we may be taking too much credit for eliminating the pain, as treated during those short attacks, since the pain in 41.6 per cent of patients lasts less than one hour, even if untreated.

INCIDENCE

Eighty-two per cent of the patients in our series were females with an average age of 46.9 years and 18 per cent were males with an average age of 52.3 years. The age span ranged from 18 to 78 years, with 70 per cent of the patients being between 30 and 60 years of age. That this group represents patients who have a true chronic disease is shown by the average time of 4.6 years which had elapsed between the onset of symptoms and cholecystectomy. Sex incidence in relation to disease duration had little significance since the average period of symptoms was 4.06 years for females and 4.7 years for males.

An analysis (table I) of the surgical pathology reports of specimens revealed the following data: chronic cholecystitis with stones, 187 cases or 88.6 per cent; chronic cholecystitis without stones, 14 cases or 6.6 per cent; normal gallbladder without stone, 4 cases or 1.9 per cent; hydrops of the gallbladder, 3 cases or 1.4 per cent, 1 of which was due to metastatic carcinoma from an unknown primary; primary carcinoma of the gallbladder, 1 case or 0.5 per cent; mural lithiasis, 1

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TABLE I

Incidence of pathologic lesions in our series

Pathologic Diagnosis	Incidence
1. Chronic cholecystitis with cholelithiasis	88.6%
2. Chronic cholecystitis without cholelithiasis	6.6%
3. Normal gallbladder without stones	1.9%
4. Hydrops of the gallbladder	1.4%
5. Primary carcinoma of the gallbladder	0.5%
6. Mural lithiasis	0.5%
7. Hypertrophy of the gallbladder with diverticulum	0.5%

case or 0.5 per cent; and hypertrophy of the gallbladder with diverticulum, 1 case or 0.5 per cent.

MANIFESTATIONS

It generally is accepted that the diagnosis of chronic cholecystitis is made by the history plus the Graham-Cole test. Physical findings are minimal or absent except during acute attacks.

The hazards of cholecystectomy, as evidenced by the copious literature on common duct strictures and their repair, lead one to the conclusion that a diagnosis resulting in removal of the gallbladder must be made with care and with the assurance that such an operation will relieve the patient's symptoms. Many patients, particularly females, who have gallbladder disease without significant pain will have associated vague dyspepsia, eructations, flatulence and chronic constipation, a syndrome referred to as *gallbladder dyspepsia* by Littler and Ellis.[1] In our follow-up studies, less than half of this group obtained relief from these complaints by having their gallbladders removed. We believe, therefore, that it is a mistake to promise relief from vague gastro-intestinal symptoms following cholecystectomy. If one demands pain as a primary symptom his successful results will be higher and the patient will be spared needless surgery. However, most clinicians now recommend cholecystectomy in young and middle age individuals (under 50 or 55 years) without symptoms, but with positive cholecystograms, to prevent complications which are so apt to develop over a period of many years.

Pain. Manifestations are described in textbooks as consisting of a severe right upper quadrant pain, colicky in nature, sudden in onset and cessation, radiating to the right scapular area and often leaving behind a sense of right upper quadrant soreness. If the duration of attack is mentioned it usually is stated to be a matter of a few hours to a few days.

As stated previously certain of these classic symptoms are at variance with our findings, the most pronounced being the duration of individual attacks. Table II shows that in 41.6 per cent of our patients these attacks lasted less than 1 hour, and in 30.5 per cent they lasted less than 30 minutes. Of the 58.4 per

cent of patients whose pain exceeded 1 hour in duration, 85.7 per cent stated that their pain lasted less than 24 hours. Thus, we believe that a more accurate description of the duration of a single episode of pain in chronic gallbladder disease is *one lasting from a few minutes to a few hours and uncommonly exceeding 1 day*. Furthermore, we would tend to doubt the diagnosis of chronic cholecystitis if the patient stated that his pain was unrelenting for several days, unless he had a hydrops or empyema.

Colicky pain is the expression most frequently used in describing the type of pain in gallbladder disease. We believe the term *colicky* is frequently misused, as is the term *cramping* when applied to any lower abdominal pain. By improper employment, both have become synonymous with pain from a certain site rather than a true intermittent pain which comes and goes in waves, each crest and valley being of short duration. In our series, 43 per cent of the patients stated that their pain was continuous and unabating throughout the entire episode rather than intermittent or colicky. We believe this fact is not fully appreciated. It is important, then, for the patient to differentiate the duration of each wave of pain from the entire period of discomfort. Also, 40 per cent of our patients with pain stated it was dull and aching, while 60 per cent had a sharp stabbing pain. The severity of pain is a difficult thing to assess because of the wide variation of individual pain thresholds. Therefore, although 68 per cent of the patients stated that their pain was severe, this in itself may not be significant since gallbladder

TABLE II

Duration and type of pain with each attack

Duration of Attack	Per Cent Intermittent	Per Cent Continuous	Total %	
1. Less than 1 hr.				
0–15 min	9.7	2.8	12.5	
16–30 min	9.7	8.3	18.0	41.6
31–60 min	6.9	4.2	11.1	
2. More than 1 hr.				
1–24 hours	15.4	34.7	50.1	58.4
over 24 hours	1.4	6.9	8.3	
	43.1	56.9		

TABLE III

Site of initial pain

Site	Per Cent
1. Right upper quadrant only	45.6
2. Midepigastrium only	27.2
3. Right upper quadrant and midepigastrium	18.9
4. Other	5.3
5. None	2.9

pain may be of variable intensity. We do know, however, that pain is the most frequent symptom causing a person to seek medical attention for gallbladder disease.

The site of initial pain (table III) also is inconsistent. Onset of pain limited to the right upper quadrant only occurred in 45.6 per cent of our series, as opposed to 27.2 per cent originating in the midepigastrium only. One-third of these latter patients never had pain in the right upper quadrant at any time during their history.

Clinicians must constantly be aware of the fact that occasionally the location of pain (or its referral site) may be very atypical. Atypical location of pain occurred in 5 per cent of our patients. Diagnosis in such instances may be extremely difficult; the roentgenogram may reveal a pathologic gallbladder, but it must be remembered cholecystitis and cholelithiasis are so common that they often are associated with other diseases producing symptoms in the upper abdomen, while the gallbladder disease is asymptomatic. These patients present a perplexing problem; too frequently they are either operated upon indiscriminantly or dismissed as neurotic.

Radiation of pain was totally absent in 24.5 per cent of our patients (table IV). When radiation was present, the most frequent site was the right scapular area with 37.8 per cent. The interscapular region was second in frequency with 26.7 per cent incidence.

Jaundice. Jaundice sometime in the course of the disease, occurred in 47 pa-

TABLE IV

Site of pain radiation

Site	Per Cent
1. Right scapula	37.8
2. Between scapulas	26.7
3. Left upper quadrant	3.9
4. None	24.5
5. Other	7.1

TABLE V

Incidence of jaundice in 211 cases of chronic cholecystitis

(47 of 211 or 22.2 per cent)

	Cases	Per Cent
Total number jaundiced	47	100
Male	14	29.8
Female	33	70.2
		Stones found
Jaundiced at operation	17 (36%)	13 (76.5%)
History of jaundice—no icterus at operation	30 (64%)	7 (23.5%)

tients or 22.2 per cent of the total of the 211 patients (table V). Thirty-three (70.2 per cent) were female and 14 (29.8 per cent) were male. Of these 47 patients, 17 (36 per cent) were jaundiced at the time of operation and 30 (64 per cent) presented no clinical icterus but gave a positive history of jaundice during at least one previous attack.

Miscellaneous Symptoms. Nausea and vomiting occurred in 60 per cent of the series; these are important symptoms since vomiting, either spontaneous or induced, frequently relieves pain in gallbladder disease.

Except for jaundice, previously mentioned, the physical findings have not been very significant in the cases studied. Most of our patients have been seen in the intervals between attacks and rarely exhibit much abdominal tenderness or muscle spasm. In surveying the clinical records we have been impressed with the frequency of negative abdominal findings. However, most patients, unless the examiner is very gentle, will complain of some abdominal tenderness on palpation, particularly in the right upper quadrant.

COMMON DUCT EXPLORATIONS

Of the entire series of 211 patients, 41 (19 per cent) had common duct explorations. The indications for these explorations included: jaundice at the time of surgery, a history of jaundice, palpable stones in the common duct, multiple small stones in the gallbladder, dilation of the common duct, and palpable or visible thickening of the common duct wall. We wish to warn that a history of jaundice alone may not constitute an indication for choledochostomy because the most common type of jaundice is nonsurgical, namely virus hepatitis. The jaundice must be of the obstructive type if it is used as an indication for opening the common duct.

Twenty-five (61 per cent) of the 41 explored ducts contained stones constituting 11.9 per cent of the entire series (table VI). Of these 25 patients with choledocholithiasis, 20 (80 per cent) had been jaundiced at least once during their gallbladder disease or were jaundiced at the time of operation. Thirteen (52 per cent) were jaundiced at the time of operation. As mentioned above, 47 patients had jaundice some time during the course of their disease. The common duct was explored in 30 of these 47 patients, but stones were found in only 19. The re-

TABLE VI

Common duct explorations in 211 cases of chronic cholecystitis

(41 ducts, 19 per cent of series explored)

	Cases	Per Cent
Total explored common ducts	41	100
Contained stones	25	61
No stones	16	39
Patients with choledocholithiasis	25	100
Total jaundiced during disease	20	80
Jaundiced at operation	13	52
Jaundiced previous to operation	7	28

maining 17 ducts were not explored because there was no jaundice at operation and the common ducts were entirely normal by inspection and palpation. Thus, 40.4 per cent (19 of 47) of the patients with a history of previous jaundice or jaundice at operation had common duct stones and 59.6 per cent (28 of 47) had none.

In the group of 19 patients with a history of jaundice (previous to the time of operation) who had common duct stones on exploration, 47 per cent had right upper quadrant or midepigastric pain with no associated radiation. Forty-three per cent had radiation of pain in the area of the right scapula or between the scapulas, and 5 per cent in the left upper quadrant. Five per cent had neither primary nor radiating pain.

SUMMARY

A series of 211 consecutive patients having cholecystectomies, both with and without choledochotomy, was analyzed with primary consideration for the duration, type and location of pain in this disease. Contrary to common belief, the pain in individual attacks of chronic cholecystitis is of short duration. The attacks lasted less than 30 minutes in 30.5 per cent, less than 1 hour in 41.6 per cent and less than 24 hours in 91.7 per cent of this series. A history of unrelenting right upper quadrant pain for a period of days tends to rule out chronic cholecystitis unless a hydrops or empyema is present.

Intermittent and *continuous* are terms more applicable than the term *colicky* when describing the type of pain in chronic cholecystitis. A surprisingly large percentage (43 per cent) of our patients had a continuous type of pain whereas in the remaining 57 per cent, pain was of the intermittent type.

The pain was located in the right upper quadrant in 45.6 per cent, and in the midepigastrium only, in 27.2 per cent. It was located in both the epigastrium and right upper quadrant in 18.9 per cent and elsewhere (frequently left upper quadrant) in 5.3 per cent.

The common duct was explored in 19 per cent of the series; stones were found in 61 per cent of those explored, representing 11.9 per cent of the entire series. Only 52 per cent of patients with stones in the common duct were jaundiced at the time of operation.

We had no postoperative deaths in this series. However it is admittedly too small to allow deduction regarding mortality rates. Surveying other large series, it is evident that the mortality rate for cholecystectomy alone should be less than 1 per cent, and the mortality rate of choledochostomy plus cholecystectomy should be perhaps somewhat less than 2 per cent.

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SPONTANEOUS PERFORATION OF THE COMMON BILE DUCT

REPORT OF TWO CASES WITH RECOVERY*

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Spontaneous rupture of the common bile duct is a relatively rare condition. Reports of spontaneous rupture occurring in patients who have had no previous biliary tract surgery are very infrequent, there being only about 14 recorded cases.[1] Over twice as many cases are reported in patients having had previous biliary tract surgery[5].

About 90 per cent of cases are caused by a stone impacted in the common duct, either below or at the site of perforation.[2] In the remaining cases, the etiology is somewhat obscure. Various theories of the etiology have been advanced including, (1) reflux of pancreatic secretions with digestion of the duct wall, (2) mural thrombosis, (3) spasm of the sphincter of Oddi causing increased intraductal pressure, (4) rupture of a diverticulum or cyst of the common duct, and (5) infection of the wall of the duct.[4] No case of carcinoma at the site of a rupture has been reported.

The diagnosis is difficult to make preoperatively. Clinically the patient has a sudden onset of severe pain usually with some degree of shock. Signs of a rapidly spreading peritonitis are present. The differential diagnosis should include pancreatitis and perforated peptic ulcer.

The treatment for this condition is surgical as soon as the patient's general condition permits. Conservative therapy has resulted in a mortality rate of almost 100 per cent. The definitive procedure usually has been the removal of common duct stones, if present, and closure of the perforation with T tube drainage of the duct. The mortality rate of patients who were operated upon is around 60 per cent.[3]

CASE REPORTS

Following are 2 case reports of spontaneous rupture of the common bile duct with recovery of the patients.

Case 1. A 50 year old, poorly nourished, Latin American woman was admitted to the hospital on Feb. 21, 1954 because of severe, intermittent right upper quadrant pain radiating to the back and right shoulder of four days duration. She had had similar less severe attacks for several years. She had noted clay colored stools and a gradually increasing jaundice for the past several weeks. Nausea and vomiting had been present for three days. Further history and system review were noncontributory. There had been no previous surgery.

Physical examination. She was acutely ill but well oriented. There was evidence of dehydration and icterus of skin and sclerae. The temperature was 101.2 F., and the pulse was 100. The abdomen was slightly distended. Tenderness was generalized, but more marked

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in the right upper quadrant. Moderate rigidity and rebound tenderness were present in the right upper quadrant. Bowel sounds were hypoactive. Masses were not palpable. Rectal examination showed light yellow stool. The white blood cell count was 32,600 per cu. mm. with 92 per cent neutrophils. The hemoglobin was 7.1 Gm. per 100 cc., with a red blood cell count of 3,000,000 per cu. mm. The urine was within normal limits. The direct bilirubin was 1.5 mg. per cent, and the indirect 7.3 mg. per cent. The total bilirubin was 8.8 mg. per cent.

Four hours after admission, the temperature rose to 104 F. and the pain, rigidity, and tenderness increased. She was given intravenous fluids and blood transfusions before operation.

When the abdomen was entered, a large mass of adhesions was found in the right upper quadrant. While the adhesions were being dissected from the liver, a small abscess was entered which connected directly with the common bile duct. A stone 1 by 1 by 0.5 cm. in size was found impacted in the duct distal to the point of perforation. The gallbladder was small and contracted and lying almost posterior to the greatly dilated common duct. The gallbladder was removed. The common bile duct was very friable and inadvertently was torn near the duodenum during the dissection. The tear in the common bile duct was repaired and drained with a T tube at the site of the perforation.

Postoperatively, the patient did fairly well and the jaundice gradually disappeared. After 10 days, the total bilirubin was 1.8 mg. per cent. At three weeks a cholangiogram showed no obstruction with normal emptying into the duodenum. The T tube was removed and the patient has continued to do well.

Comment: This was a case of spontaneous rupture of the common duct in a patient who had had no previous biliary tract surgery. As in most of the reported cases, perforation was due to an impacted gallstone in the common duct. Because of the friability of the tissue and distorted anatomy, an injury to the common duct was produced during surgical exploration. This was recognized and repaired.

Case 2. A 65 year old obese, Latin American woman was admitted to the hospital on Feb. 19, 1954. On that day she suddenly developed severe upper abdominal pain which radiated to the right shoulder. She did not vomit.

There was a history of an operation upon the gallbladder in 1939. A record of this procedure was not available.

The patient was obviously in severe distress. The blood pressure was 130/80, the pulse was 100, and the temperature 101 F. The abdomen was obese. There was a long right rectus scar and large incisional hernia. Distention was marked and there was generalized tenderness and rebound tenderness. Peristalsis was not heard. Brownish colored fecal material was present in the rectum.

The hemoglobin was 13.6 Gm. per 100 cc. The red blood count was 5,104,000 per cu. mm. The white blood count was 8,700 per cu. mm. with 84 per cent neutrophils. The urine showed 2-plus albumin, but otherwise was normal. The total serum bilirubin was 1.6 mg. per cent. The serum amylase was 101 mg. per cent (normal 60–130 mg. per cent).

The patient was hydrated with intravenous fluids and an exploratory operation was done. When the peritoneal cavity was opened, a peritonitis was found with free bile present. Moderate adhesions were found in the right upper quadrant. The gallbladder was not present. An opening 1 cm. in length was found in the supraduodenal portion of the common duct on the anterior wall through which bile was escaping into the peritoneal cavity. The duct, which was not dilated, was explored through this opening and no obstructive lesion found. The pancreas was thought to be normal. A T tube was placed in the duct at the site of rupture and the duct was closed around it with no. 00 chromic catgut. The abdomen was closed and drained.

The patient had a rather stormy postoperative course with an episode of right lower

lobe atelectasis. She was treated with antibiotics and general supportive measures and gradually improved. Cholangiograms were done on two occasions and stones were not demonstrated. A narrowing near the sphincter of Oddi was present and was interpreted as being due to spasm. On the sixteenth hospital day the T tube was inadvertently removed. The patient's condition continued to improve and she was dismissed on the twenty-sixth hospital day and has remained in good health.

Comment: The etiology of this ruptured common duct is obscure. The duct was not dilated and stones were not present. It is unfortunate that records of her previous surgery could not be obtained.

SUMMARY

Two cases of an evidently rather rare condition are reported.

Case 1 was a patient without previous surgery who had a ruptured common duct due to a distally impacted stone.

Case 2 was a patient who had a rupture of the common bile duct following cholecystectomy 15 years before. The etiology of the perforation in this case is unknown.

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TORSION OF THE GALLBLADDER WITH ASSOCIATED ACUTE GANGRENOUS CHOLECYSTITIS

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In an examination of the American medical literature, it was surprising to find how seldom torsion of the gallbladder was mentioned. A careful study by Haines and Kane[4] of torsion of the gallbladder in 1948, revealed that there were only 76 cases reported to date in the American literature. In reviewing files at the Library of Congress in Washington, D. C., it was interesting to note the frequency with which this entity is mentioned in the foreign literature, particularly German. The general consensus of the American authors on this subject is well summarized by McLean and Ellis[7] who say, "It is seldom that one observer will see more than one case in his lifetime, and therefore possibilities of recognition are slight."

Although torsion of the gallbladder rarely is seen and even more rarely diagnosed preoperatively, it should be suspected in visceroptotic individuals of the female sex between the ages of 60 and 80 years. The average age range of patients are reported between the years of 11 and 83; however, in the French literature, a case is reported by Dabadie[1] in a 13 month old infant. It is estimated that less than 8 per cent of the cases occur in the male and such a case is reported by Forty[2] in a British medical journal. It is expected that the incidence of this condition will be noted more frequently in the future with the advent of an increasingly aging population.

Because of the apparent rarity of this condition, yet with the possibility of its increasing frequency with an aging population, and because the condition is undiagnosed in practically all patients prior to surgery, a brief survey and case report are presented.

ETIOLOGY

In 1932, an article by Meeker and Lisenby[6] entitled "Ptosis and Torsion of the Gallbladder" clearly reviews the salient features of the condition. It is estimated that 20 per cent of all patients have a ptotic type of gallbladder. The important factor in the production of torsion is a long gallbladder mesentery which permits rotation. With loss of elasticity of supporting structures in the older age group, it is more feasible to understand that peristaltic motion of the transverse colon acting upon the gallbladder in the area could cause torsion of the gallbladder in those patients with a long mesentery allowing rotation. Rotation occurs with the pivot at the cystic duct. It would appear that incomplete torsion of the gallbladder with partially obstructed circulation could occur frequently in these individuals, but only when circulation has been completely obstructed does ischemia with subsequent gangrene give rise to sufficient abdominal symptoms that the patient consults a physician.

PATHOLOGY

Pathologic findings of torsion of the gallbladder are similar to those noted in the case cited below. The gallbladder may be slightly enlarged; rarely contains stones, and is tense and distended. The walls externally are smooth and thickened, purplish-black in color, and present evidence of gangrenous changes depending upon the duration of the ischemic process. The lumen usually contains a thick, dark, semi-fluid, bloody-appearing material with or without evidence of biliary tinge. The gallbladder may be rotated 180 to 360 degrees. It is surprising how little inflammation occurs proximal to the point of rotation of the mesentery if prompt surgery is instituted.

SIGNS AND SYMPTOMS

The signs and symptoms of torsion of the gallbladder are not uniform. They vary from that simulating acute appendicitis to dissecting aneurysm of the aorta, or perforated peptic ulcer as in a case cited by Gowland.[3] On rare occasions there may be, not only a torsion of the gallbladder, but acute appendicitis; such cases are cited by Husband and Schmitt.[5] Occasionally the onset simulates biliary colic with vomiting noted frequently. A palpable tumor in the region of the gallbladder usually appears within two hours after onset of symptoms and the mass may be tender with evidence of rigidity in the right upper quadrant. Chills and fever often are lacking. Flatulence and indigestion are infrequent in such patients. Incomplete torsion of the gallbladder may simulate acute cholecystitis and the patient might give a past history suggestive of such. Cholelithiasis rarely is associated with this condition.

The most important feature in diagnosing torsion of the gallbladder is a high index of suspicion of the condition. It occurs most frequently in the aging female of the visceroptotic type, having evidence of an acute abdominal disease. Laboratory studies, with the exception of the blood count showing evidence of a leukocytosis and elevated polymorphonuclear leukocyte count, are important only in the negative sense. An abdominal roentgenogram is not diagnostic. Peritonitis develops if the patient is untreated and either walling-off or perforation of the gallbladder will occur. It has been reported that the mortality rate is 100 per cent in patients who are not treated surgically.

PROGNOSIS

The outlook for cases of torsion of the gallbladder is excellent if the patient is operated upon early and the general condition is not too hazardous. The ease of the surgical procedure contributes to the excellent prognosis.

CASE REPORT

A 71 year old white woman appeared in the office Sept. 26, 1953, with abdominal pain. Thirty-six hours previous, the patient had an acute abdominal pain, minimal nausea and no vomiting. She took several enemas and an Epsom salt laxative without relief of her condition. Except for constipation, she had had no other gastrointestinal or abdominal symptoms and no previous abdominal surgery.

The patient was admitted promptly to the Lake Charles Memorial Hospital and physical

examination at that time showed a small, senile, 66 pound woman in severe pain. Abdominal examination showed no scars but an extremely tender abdomen with diffuse rigidity, more particularly in the right lower quadrant, and definite rebound tenderness throughout the abdomen. A positive Murphy's sign was present; peristalsis was audible. The pelvic examination was not unusual for this senile patient. A moderate degree of cul-de-sac tenderness on the right side was found. The impression at the time was an acute abdominal disease, probably acute gangrenous appendicitis. Laboratory reports showed a normal urinalysis with a hematologic report as follows: Red blood cells, 3,470,000 per cu. mm.; hemoglobin, 11 Gm., 66 per cent; white blood cells, 15,100 per cu. mm.; 88 per cent segmented polymorphonuclear leukocytes, and 12 per cent lymphocytes.

Abdominal exploration was done on the evening of Sept. 26, 1953, and there appeared a torsion of the cystic duct and the gallbladder mesentery over a 360 degree angle. Cholecystectomy was done easily. She had an uneventful recovery and was discharged from the hospital on the fifth postoperative day.

The pathologist's report indicated the gallbladder was intact, measuring 7 cm. in length, 4.5 cm. in width, 1 cm. in thickness. The organ was a dark grayish-brown color and the serosa thickened. On sectioning the gallbladder, the lumen contained a thick, very dark, red semi-fluid and bloody-appearing material. The mucosa was much discolored and details were lost. Sections from the gallbladder wall showed one general picture characterized by tissue necrosis in mass with infiltration of all coats by red blood cell pigment, few leukocytes, and fibrin. Only a few fairly well preserved blood vessels containing clotted red blood cells and a few small islands of well-defined columnar cells remained identifying gallbladder structure. No malignancy or suggestion of stone formation was found. Final Pathologic Diagnosis: Acute hemorrhagic and necrotic cholecystitis due to sudden deprivation of blood supply.

The patient was seen in the office 12 days postoperatively and some of the cotton sutures were removed from the skin wound. On Oct. 15, 1953, 19 days postoperatively, the remaining sutures were removed. The wound had healed well for this senile patient. The patient has had no further abdominal pain or discomfort and has been eating well. She was known to be alive and doing nicely as regards her abdominal condition in April, 1955.

SUMMARY AND CONCLUSIONS

A case of torsion of the gallbladder is presented with a review of the interesting features, incidence, etiology, pathology, signs and symptoms, surgical therapy, and prognosis. It is important to suspect torsion of the gallbladder in the aged, visceroptotic woman who presents herself with sudden onset of abdominal pain and evidence of peritonitis principally centered in the right upper quadrant.

Torsion of the gallbladder rarely is diagnosed preoperatively.

If torsion of the gallbladder is diagnosed as acute cholecystitis and a waiting policy adopted, death may ensue from perforation and peritonitis.

The importance of early operation is emphasized.

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GIANT CARTILAGINOUS CHEST WALL TUMOR

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Today surgeons rarely encounter visible tumors of massive proportions. Improved medical care, mass roentgenologic studies, and awakened public interest in cancer have led to the earlier examination and treatment of abnormal masses. This case of a giant chondromyxoma of the anterior chest wall is of interest because of the tremendous size of the tumor, its type, location, and rate of growth. That the tumor was permitted to become so large suggests that the benefits and safety of modern thoracic surgery are not yet fully appreciated.

CASE REPORT

F. E. This 60 year old white woman was hospitalized at Emergency Hospital, Washington, D. C., on Oct. 8, 1952, for removal of a giant chest wall tumor. She always had been in good general health except for the tumor which she said had been present approximately 20 years. The mass first became noticeable on the anterior chest wall just above the left breast about a year after she had fallen and fractured a rib in that area. She consulted her family doctor when the mass first appeared, but was told nothing should be done. The tumor grew slowly, gradually increasing in size, although it produced no symptoms. As it continued to grow, she sought other medical advice but was given essentially the same answer. The size of the tumor caused her considerable inconvenience and embarrassment and even necessitated specially tailored clothes to hide it from view. As the growth reached mammoth proportions, she repeatedly requested operative removal, but she was told that operation would be too dangerous and that her chances of surviving operation were poor. The patient finally came for help on the advice of a friendly layman.

Physical examination on admission to the hospital revealed a well developed, well nourished, 60 year old woman with a tremendous tumor of the left anterior chest wall (fig. 1). The mass was somewhat smaller than a basketball, but larger than a football. It presented in the left anterior chest region and appeared to be supramammary in origin. Some observers thought that it arose from the left breast but most believed that it was entirely separate and arose from the chest wall. The tumor extended medially to the right of the midline, laterally into the axilla, and superiorly sufficiently high that she rested her chin on it. The lower margin of the mass fused with the left breast which was pulled upward and formed the inferior border of the tumor. The skin overlying the tumor was stretched tight. The mass itself was rubbery, irregular, and noncystic, and was firmly attached to the deep structures of the anterior chest wall. There was no localized nor generalized lymphadenopathy. The position of the heart could not be determined. Breath sounds could be heard only posteriorly on auscultation of the left chest.

Roentgenographic examination of the chest showed a solid opaque mass obscuring the left lung field (fig. 2). It measured 30 cm. in its transverse diameter. The right lung field was clear. Two rounded masses were visible in the anterior mediastinum and were thought to represent intrathoracic extensions of the external tumor. There were a few calcified areas in the center of the extrathoracic tumor. The ribs appeared normal and the interpretation of the roentgenologic studies was "a large benign tumor arising from the left breast or the soft tissues of the left chest wall."

From the Department of Surgery of the Johns Hopkins University School of Medicine and the Johns Hopkins Hospital.

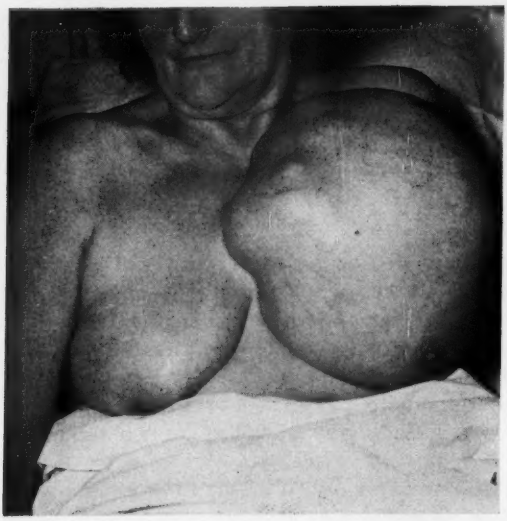


FIG. 1. Anteroposterior preoperative photograph showing the tumor pulling the left breast upward and fusing with it. The mass extends well past the mid line.

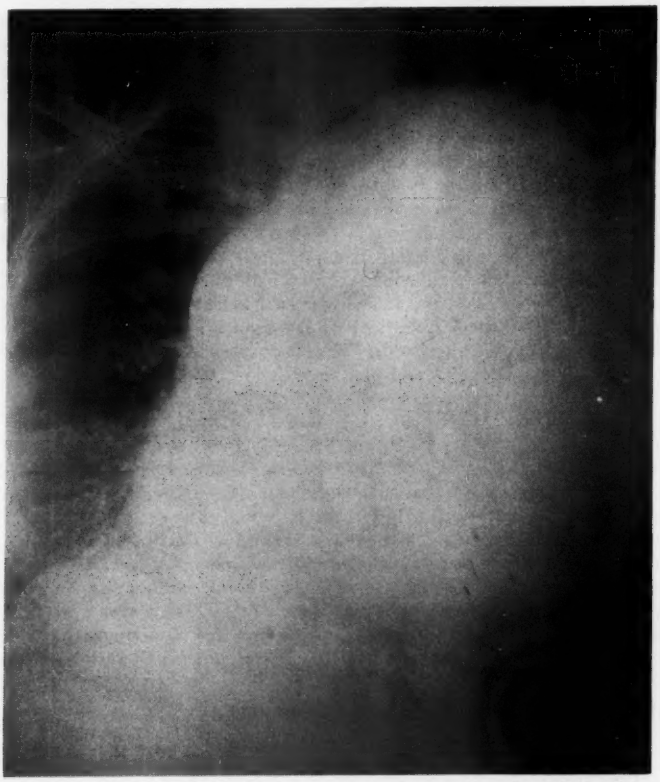


FIG. 2. Preoperative anteroposterior roentgenogram revealing the left hemithorax completely obscured by the tumor. The calcification is not well demonstrated but could be visualized on the original film.

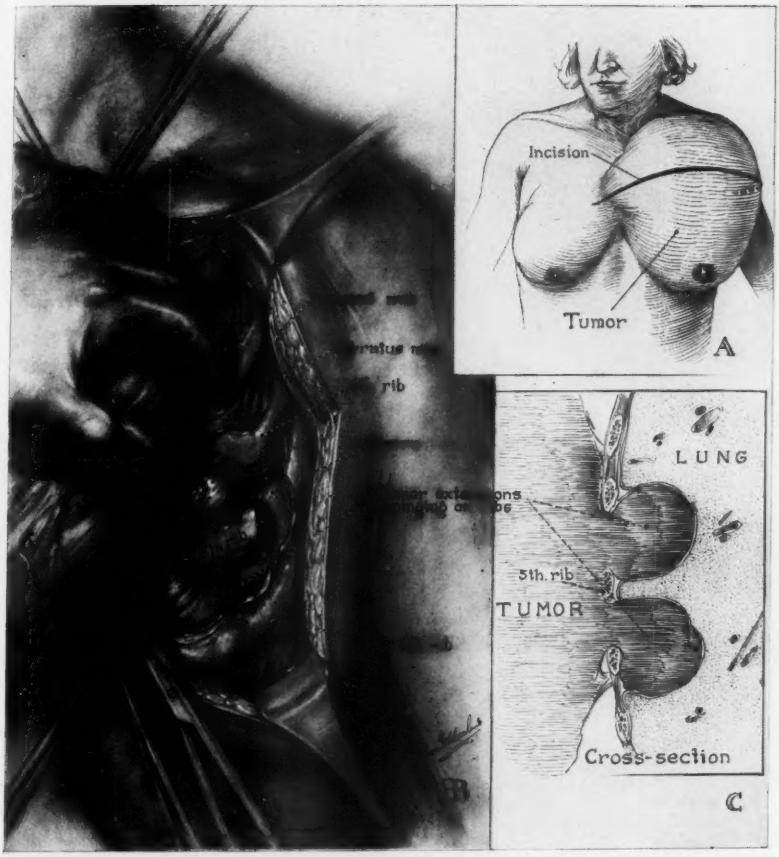


FIG. 3. Sketch of operation. A. The position of the incision in relation to the tumor is illustrated. B. The fifth rib has been divided in the anterior axillary line and the tumor is being mobilized medially in order to demonstrate the intrathoracic extensions. The fourth rib is pushed upward and the sixth rib downward. C. The intrathoracic but extrapleural tumor extensions are illustrated on cross section.

On October 9, she was operated upon under gas, oxygen, and ether intratracheal anesthesia. A transverse skin incision was made over the midportion of the tumor extending from the midline to the axilla (fig. 3). The pectoral muscles were found to be exceedingly thin but were not adherent to the mass. They were divided and the anterior surface of the tumor came into view. It was found to be very nodular but encased in a thick capsule. There were no adhesions or evidence of extension to the surrounding soft tissues. The only point of attachment to the chest wall was through a relatively narrow base at the costochondral junction of the fifth rib. It was decided that the tumor had arisen from this point and extended up in a mushroom type of growth. Block resection of this area was decided upon, and the involved rib was divided in the axilla well lateral to the left margin of the mass. Then, the tumor was mobilized medially together with segments of intercostal muscles

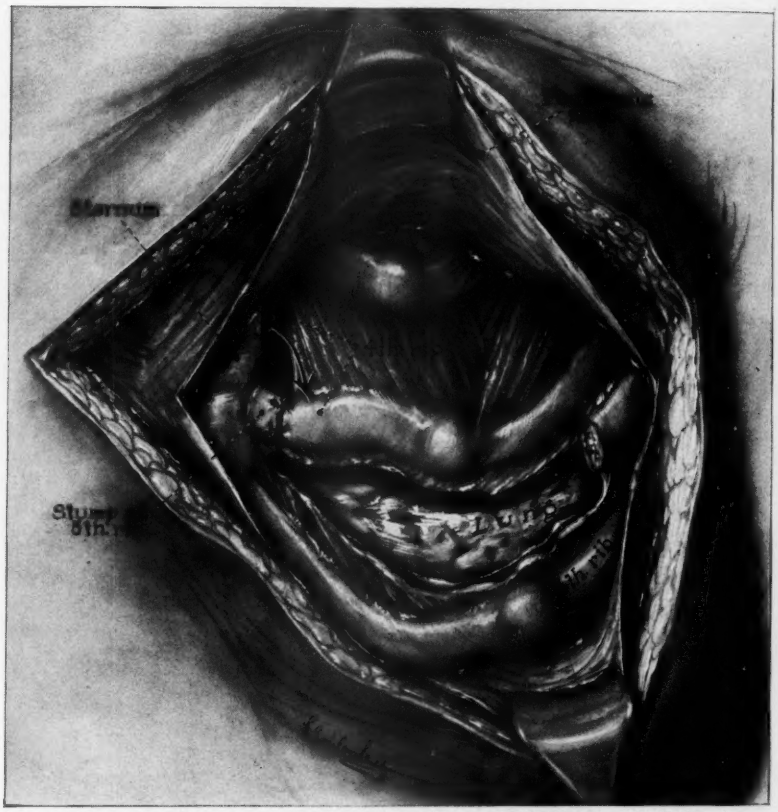


FIG. 4. Operation. The tumor has been removed as a block. The fourth rib has been divided, mobilized, and reattached to the sternal stump of the fifth rib.

and pleura. Two intrathoracic but extrapleural tumor extensions, approximately 4 by 5 cm. in size, on the undersurface of the mass then were visualized (fig. 3). They were located under and between the fourth and fifth ribs and extended inward through the intercostal spaces. The fourth rib had been pushed upward and the sixth rib forced downward. The fifth rib was divided as near the sternum as possible, and the entire specimen removed in one block (fig. 3).

This left a defect in the anterior chest wall approximately 7.5 by 18 cm. in size. This was closed by dividing the costal cartilage of the fourth rib at its sternal attachment, then this rib and its intercostal bundle were brought down and sutured to the sternal stump of the resected fifth rib (fig. 4). This served as a bridge across the midportion of the chest wall defect. Over this the pectoral muscles were sutured in a two layer, imbricating-type closure. The left breast was mobilized superiorly and sutured into position to reinforce the region of the operative defect (fig. 5). The excess of subcutaneous tissue and skin was excised. The lung was expanded, the wound was closed without drainage, and a large pressure dressing was applied.

The pathologic report was benign chondromyxoma arising from the cartilage of the fifth rib (figs. 6, 7). The two intrathoracic masses were simply lobulated extensions of the main

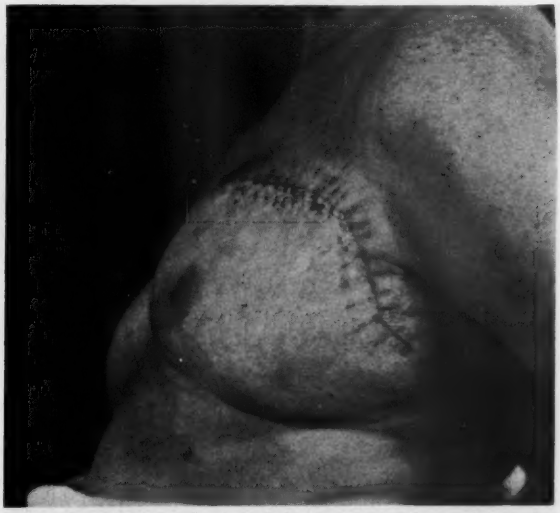


FIG. 5. Postoperative photograph. A satisfactory cosmetic result has been obtained

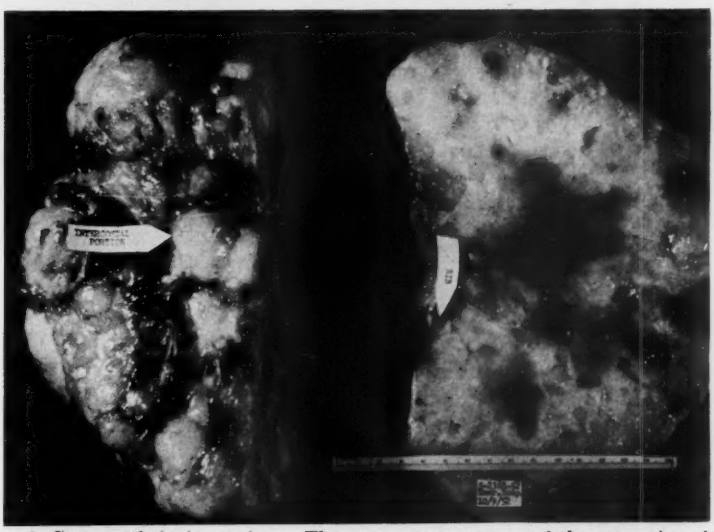
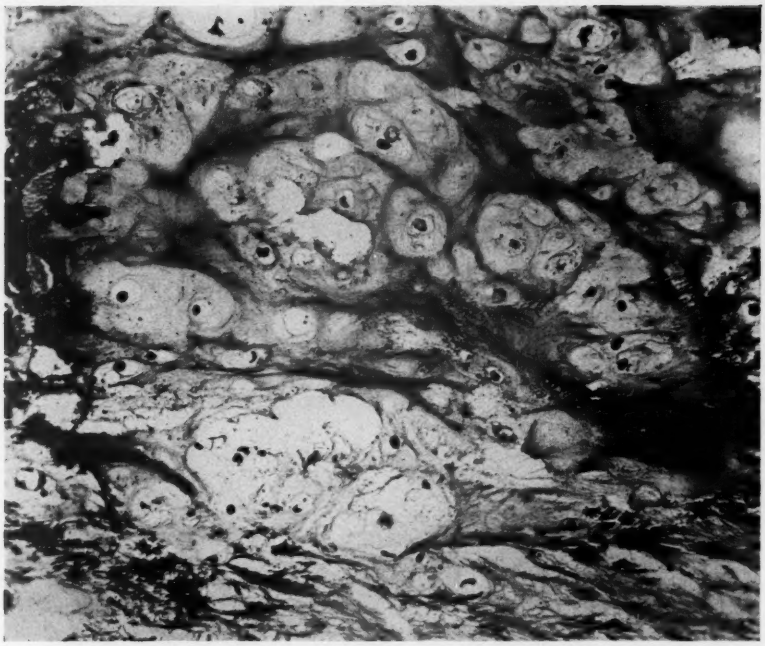


FIG. 6. Gross pathologic specimen. The myxomatous nature of the tumor is quite apparent and areas of necrosis are easily visualized. A portion of the rib and the intrathoracic extensions are labeled.

tumor. At no point was there histologic evidence that the tumor had extended past the restraining capsule.

The patient tolerated the operative procedure well. Her postoperative course was uneventful. She was ambulatory on her first postoperative day and left the hospital eight days following operation. A satisfactory cosmetic result was obtained. She has gotten



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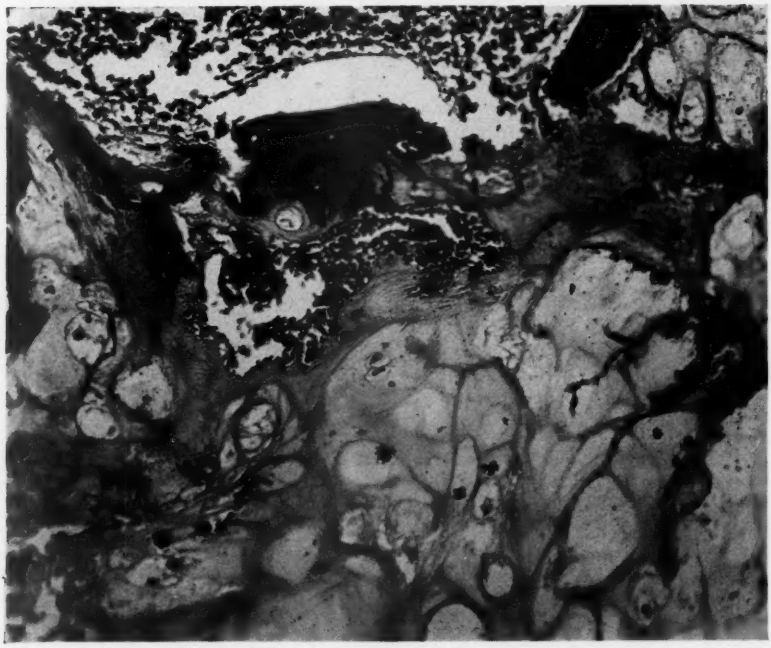

B

FIG. 7. Photomicrographs × 150. Hyperplastic lacuna are seen throughout the sections. Several mitoses are clearly visible in figure B. Tissue degeneration and myxomatous changes are marked. In the upper part of figure B normal bone and marrow can be seen. Flecks of calcification appear at the lower edge of figure A.

along well during the ensuing years. On Feb. 13, 1955 she was in good health and there was no evidence of a recurrence.

DISCUSSION

Primary chest wall tumors are much less common than are metastatic lesions. The ribs frequently are involved in the metastases of many neoplasms. Most tumors of the chest wall are malignant. Benign tumors do occur and the chondroma is the most common of these. Different stages of the same pathologic process may be represented by the presence of varying amounts of myxomatous or osseous elements; thus, the name may be changed from chondroma to chondromyxoma or osteochondroma. All cartilaginous tumors are considered to be potentially malignant although they may appear histologically benign. Tumors which grossly and histologically have been considered benign have recurred following removal and some have even produced distant metastases.[1-4, 7, 8, 11, 13, 15-18, 22, 25-27, 29, 30-37, 39-41, 43-49, 52]

Primary rib tumors present the same clinical characteristics found in similar tumors occurring elsewhere in the body.[14, 28, 38] The exact preoperative diagnosis of a chest wall tumor may be an exceedingly difficult problem. Most of these lesions are found on the anterior chest wall and the costochondral junction is a favorite site. Often, there is an incident of trauma to the region of the tumor, but the etiologic import of this finding has never been determined although it appears to be more frequent than simple coincidence.[14, 18, 19, 23] Pain and a mass are the cardinal symptoms. The pain is dull and usually appears first. Sometimes the pain occurs at the tumor site before the mass is palpable or even visible on roentgenologic examination. Occasionally the mass will be discovered before pain has occurred. This is particularly true if the tumor is deep or grows intrathoracically. As the tumor grows, the pain becomes more localized. Sharp, severe pain, pleuritic pain, fever, or pleural effusion are considered late manifestations of malignancy. Roentgenologic examination is the most important single study, and if metastatic lesions are to be ruled out, a skeletal survey may be indicated. Laminograms are particularly helpful in distinguishing rib involvement from intrathoracic or mediastinal growths. Occasionally pneumothorax or fluoroscopy are employed as diagnostic aids, but usually these are unnecessary. Needle aspiration biopsy is to be condemned, and tissue resection as a part of the exploration seems preferable.

The differential diagnosis should include metastatic lesions, tuberculosis, osteomyelitis, syphilis, mediastinal neurogenic tumors, intercostal nerve tumors, inflammatory lesions, fibrous dysplasia, hemangioma, myeloma, Ewing's tumor, or primary pleural lesions such as a localized empyema or abscess, aneurysm, or chest wall involvement from a bronchiogenic carcinoma.[5, 6, 9, 12, 14, 18, 19, 21, 24, 38] Pyogenic infections or trauma probably cause the most confusion. Fractures which heal poorly or a hematoma which remains unresolved should be regarded with suspicion and there is no more dangerous attitude than that of *watchful waiting*.

Burford states that, "No group of tumors has in general been more badly man-

aged."[9] The fear of consequences of radical resection of a segment of the entire chest wall seems responsible for most of the recurrences.[6] Early complete removal is the only treatment of choice. The frequency with which benign tumors become malignant is the most pressing indication for early removal. Neglected tumors, even if benign, may become so large that their removal is rendered unnecessarily hazardous.[19, 20, 38] The best opportunity for eradication of the tumor is at the first operation, and radical resection is urged. Incomplete removal of a cartilaginous tumor seems to stimulate the growth of the remaining tissue. Dorner[14] and others[19, 38] feel resection should include removal of the entire involved rib, periosteum, the underlying pleura and adjacent muscle such as the intercostals, and adjacent chest wall or diaphragm. If the tumor is known to be malignant, a wide block resection is done. The recurrence rate is exceedingly high unless this is accomplished. It is impossible to tell whether or not a benign tumor has undergone malignant changes, but suggestive findings are those of sudden increase in the size of the tumor or the sudden development of pain in a previously recognized mass.

Repair of chest wall defects following operation may be accomplished if plastic procedures on nearby tissues, such as the breast, ribs, muscle flaps or diaphragm, are combined with a prosthesis like tantalum mesh.[10, 42, 50, 57]

Radiation therapy has no place in the treatment of these lesions.

SUMMARY

A giant chondromyxoma of the chest wall is reported. Some of the characteristics of cartilaginous tumors and the accepted method of treatment are discussed.

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PERFORATION OF THE COLON AFTER BARIUM ENEMA AND AIR CONTRAST STUDIES

REPORT OF A CASE*

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Reports of traumatic perforation of the colon due to nonpenetrating abdominal injuries, such as compressed air introduced through the rectum, enemas and proctoclyses, and foreign bodies, either ingested or introduced directly into the rectum, have appeared in the medical literature with increasing frequency since Stone[16] reported the first case of pneumatic perforation of the colon in 1904. Perforation of the colon by barium enema, with or without air contrast studies probably is not as rare an event as might be concluded from the paucity of reports of such incidents in the medical literature.

REVIEW OF THE LITERATURE

Kaulich[9] (1930) reported a case of perforation of the sigmoid during a repeat barium enema given 27 days after the original, following which an impaction of barium occurred; presumably there was an ulcer at the site of perforation. Himmelman[7] (1932) recorded cases of perforation of carcinomatous stenotic colons and recti following barium meals. Scheidt[14] described perforation of a carcinomatous colon during a repeat barium enema given 10 days after the original. Kleinsasser and Warshaw[10] reported a case of perforation of a sigmoid with diverticulitis during a barium enema. Isaacs[8] reported 2 cases of sigmoidal perforation during barium enema; 1 occurred in a patient who had carcinoma of the sigmoid; the other occurred in a patient who had had a sigmoidoscopic examination and the sigmoid evidently was perforated by that examination on the day preceding the barium enema. Berk[2] reported a case of perforation of a carcinomatous colon during an attempted barium enema through a colostomy stoma. Serjeant and Raymond[15] recorded a case of perforation of an apparently normal colon 24 hours after a barium meal.

PATHOLOGIC PHYSIOLOGY

As might be expected, most of the reports of perforation of the colon by barium enema have been described as occurring in diseased colons. The diseases commonly associated with nontraumatic perforation of the colon are diverticulitis, carcinoma, ulcerative colitis, amebiasis, ulcerative tuberculo-enteritis, and foreign bodies. Rankin, Bargen, and Buie stated that perforation occurred in 3.4 per cent of 647 patients who had ulcerative colitis, all of whom had far-advanced disease. Perforation now is believed to be uncommon in tuberculo-enteritis. Amebiasis has been repeatedly incriminated as a cause of perforation of the

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colon,[4, 5, 6, 12, 17] but this author was unable to find a report of a case of perforation of a colon infected with amebiasis occurring during a barium enema.

In order that the mechanisms and pathologic physiology of perforation of the colon may be better understood, a review of some of the experimental work which has been done is in order. Quenu[13] (1882) is credited with some of the first experimental work in pneumatic rupture of the colon and intestines; he found that the intestine would bear pressure equal to 50 to 60 cm. of mercury without rupture, but that when the pressure was increased to 70 cm. of mercury the intestine would rupture in several places, and he assumed that similar pressures would be required to rupture the intestine within the body. Andrews[1] (1911) tested ox, dog, and human intestine and never found one capable of bearing more than 12.5 pounds of pressure per square inch (65 cm. of mercury); the average was between 7 and 8 pounds per square inch (36 to 41 cm. of mercury). Senn, cited by Burt,[3] produced rupture in a bowel diseased with enteritis at 1.5 pounds per square inch (7.8 cm. of mercury).

Burt (1931) reviewed the literature of pneumatic rupture of the colon; reported 3 additional cases, and described experiments which he did upon the alimentary tracts of 18 human beings at autopsy, 7 males and 11 females, ranging from 5½ months to 82 years of age. He found that the average pressure required to rupture the outer two coats of the intestine was 3.47 pounds per square inch (18 cm. of mercury), and pressure required to perforate completely through the mucosa, which perforated last, was 4.07 pounds per square inch (21 cm. of mercury). No section of intestine was found to support a pressure greater than 11.59 pounds per square inch (60 cm. of mercury), which was borne by the sigmoid of an 11 year old child. Intestinal segments of children under 12 years of age were found to support higher pressures before rupturing than corresponding segments of adult intestine. The highest pressure supported by a segment of adult intestine was 8.36 pounds per square inch (43 cm. of mercury). Segments of the alimentary tract supported the highest pressures in the following order: rectum, sigmoid, ileum, esophagus, jejunum, transverse colon, cecum, and stomach. Rupture usually occurred along the antimesenteric side in a longitudinal direction. In ruptures occurring along the mesentery, the escaping gas dissected up along the mesentery or around the subserosal circumference of the intestine and then perforated in one or more fine points, commonly along the side of the bowel, and at times considerable distance from the original perforation. Introduction of air twice as fast resulted in a lowering of the pressure required for perforation. A pressure of 4.07 pounds per square inch (21 cm. of mercury) was required to rupture the bowel *in vivo*.

Wangensteen[18] stated that the greatest stress is borne by the intestine with the largest diameter, that is, the tension on the wall of the intestine increases in proportion to the diameter of the intestine, according to the following formula:

$$T = \pi DP$$

T is equal to the tension on the intestinal surface in centimeters of water per square centimeter, D is equal to the diameter of the intestine in centimeters, P

is equal to the intraenteric pressure in centimeters of water, and π is equal to 3.14. This may account for the rupturing of the larger segments of the alimentary tract, e.g., stomach and cecum, at lower intraluminal pressures than segments of smaller diameter, and also it may account for the higher pressures required to rupture segments of intestine in children.

Although Burt found the sigmoid to be the second strongest segment of alimentary tract which he tested, the sigmoid appears to be the most commonly ruptured in clinical experience, particularly in compressed air injuries and other injuries resulting from sudden increases per rectum of the intraluminal pressure of the bowel. Andrews called attention to the angulation of the sigmoid and to the fact that a column of gas or fluid suddenly introduced through the anus is momentarily trapped by this angulation; fecal masses, if present, also hinder passage of the gas column; the rectum is well supported by surrounding structures, except at the rectosigmoid, and the force thus is transmitted to the sigmoid. The ileocecal valve is another factor in confining the increase in pressure to the colon. In 50 per cent of the cases studied by Lapp,[11] the valve was competent, and the increased intracecal pressure draws the two frenula of the valve together, preventing retrogression of the gas or liquid.

Kleinsasser and Warshaw calculated the pressure of the barium suspension which caused perforation in their patient and found it to be 7.66 cm of mercury at the end of the tube (height of column 3 feet, specific gravity of barium suspension 1.070). This pressure is considerably less than the average pressure required for perforation in Burt's experiments, but it compares favorably to the results obtained by Senn in diseased bowel. They also postulate that the added weight of the barium may have contributed to the perforation. They injected dogs intraperitoneally with barium sulfate suspensions (20 to 150 ml.), mixtures of barium sulfate and unsterile stool, and unsterile, sterilized, and filtered stool suspensions. All dogs injected with the barium alone survived, except 1 which died two months later of other causes, and no incidence of significant peritoneal adhesions developed except in 1 dog. All dogs injected with barium and stool mixtures died within 48 hours. Dogs injected with unsterile stool all died, and cultures of their blood grew *E. coli*. No reaction occurred in dogs injected with sterile or filtered stool.

DIAGNOSIS

In making the diagnosis of perforation of the colon by barium enema, an alert radiologist may observe the accident as it occurs under the fluoroscope in certain instances. Isaacs describes a particular sign for prompt recognition of perforation of the sigmoid by barium enema. The barium may collect in the space between the descending and sigmoid colon and the lateral abdominal wall; the difference in density between the free barium and that within the colon enables it to be visualized along the opaque colon with the free barium showing a smooth left border where it is delimited by the parietal peritoneum; this may appear as a linear streak of density external to the bowel and distinctly separated from but parallel to the descending of sigmoid colon. Pneumoperitoneum, pneumome-

diastinum, and subcutaneous emphysema may be present and recognizable both clinically and by roentgenogram.

Clinical signs of perforation of the colon may include a subnormal temperature which will rise as peritonitis develops, rapid, shallow, thoracic respirations, weak and rapid heart action, various degrees of shock, distended tympanitic abdomen, elevated diaphragm, intensely protruding hernias if such are present, and a positive Hammond's sign or mediastinal crunch. At operation, gas or fluid, which usually is malodorous, may escape upon incising the peritoneum; emphysema of the omentum or retroperitoneal tissues may be present; an incompletely ruptured bowel probably will remain distended, whereas one that has been completely perforated will be of normal calibre. If the perforation is minute and not easily detected, an area of subserosal hemorrhage in the area of the perforation may give a clue to its location.

TREATMENT

Immediate surgical intervention is imperative in cases of suspected perforation of the colon, whatever the cause. The surgical procedure may be varied to suit the needs of the injury. In cases of pneumoperitoneum, decompression should be done preliminary to laparotomy in order to relieve shock; facilitate respiration, and prevent tearing of the tissues when the peritoneum is opened. If barium or other material is found free in the peritoneal cavity, peritoneal lavage with removal of the foreign material should be done. Enemas of any kind in cases of suspected perforation of the colon obviously are contraindicated.

MORTALITY

Mortality and morbidity rates of patients with perforated colons are high. The number of cases of perforation of the colon by barium enema appears to be insufficient to draw any accurate conclusions regarding mortality rates. Lapp reviewed 78 cases of pneumatic—*compressed air hose injury*—rupture of the colon; 10 patients were incompletely ruptured; of the 5 of these patients who were operated upon, 1 died. Thirty-eight of the 68 patients who had complete rupture were operated upon, 22 of whom survived (78 per cent mortality rate); the high mortality rate is attributed to late diagnosis or delayed operation. Every patient operated upon within the first six hours after injury survived, whereas only 1 patient who was operated upon six or more hours after injury survived. In Burt's review of 48 cases, the total mortality rate was 58.81 per cent; the operative mortality rate was 44.82 per cent, and the nonoperative mortality rate was 80 per cent.

CASE REPORT

History: M. A. T. was a 28 year old, white married woman admitted to the hospital on Feb. 16, 1953, from another hospital, with a diagnosis of moderately advanced bilateral upper lobe pulmonary tuberculosis, tuberculous enteritis, and possibly old tuberculosis of the hip. She had been a partial invalid most of her life, her history beginning at the age of 3 years when she fell and injured her right hip, following which some shortening of the right lower extremity developed. At the age of 10 years, she had measles and pertussis simul-

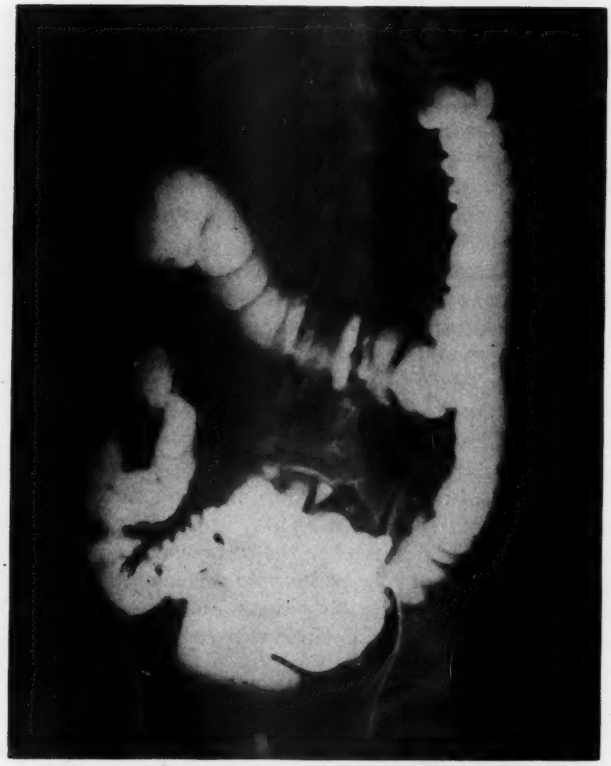

FIG. 1. Barium Enema, Dec. 17, 1952, showing marked constriction of ascending colon proximal to hepatic flexure.

taneously, and, upon recovery, she was unable to walk because of pain in the right hip, whereupon her parents were advised that she had tuberculosis of the hip. The cows from which the family received milk were tested and found to be infected with tuberculosis. She was treated by an orthopedist for her disease. At the age of 15 years, she developed severe pleurisy and was treated for tuberculosis by six months of bed rest. She recovered, except for a slight limp, and from 1943 to 1952 was relatively well; attended college on a restricted curriculum; received her degree; worked for a year as a secretary, and finally married. In October 1952, she developed severe cramping abdominal pain which lasted most of the night but which finally was relieved by codeine. On December 14, she again developed severe cramping abdominal pain for which she could obtain no relief and for which she was hospitalized. A gastrointestinal barium study and a barium enema were done and she was told that she had a partial intestinal obstruction and tuberculous peritonitis (fig. 1). She was started on a liquid diet and streptomycin and para-aminosalicylic acid therapy. Her weight dropped from 89 to 69 pounds in two months, and she then was transferred to this hospital. Here she was treated with dihydrostreptomycin, isoniazide hydrazide, and para-aminosalicylic acid for approximately 17½ months before the episode occurred which resulted in operation. She made satisfactory progress with the exception of three or four bouts of symptoms of partial intestinal obstruction during the early part of her hospitalization and an episode which occurred in March 1953, when she developed a right hemiplegia

with aphasia, which was diagnosed as a cerebrovascular accident of undetermined etiology, and from which she made a good recovery under the guidance of the Physical Medicine Department. Although reports from the referring hospital indicate that acid fast organisms had been identified in her sputum, no acid fast organisms were ever identified upon smear or culture of many sputums, gastric washings, and bronchial washings in this hospital. Barium enema was attempted upon three different occasions and showed an obstruction of the colon just proximal to the hepatic flexure, beyond which no barium could be passed. A barium progress meal indicated a nearly complete obstruction. In March 1954, after many nearly symptomless months, she complained of passing blood in her stools. She had a sigmoidoscopic examination and numerous ulcerative lesions with bleeding points were seen; apparently no microscopic study of material from these lesions was done. On July 30, 1954, it was believed that she was approaching the end of her period of maximum hospital benefit, and a repeat barium enema with air contrast studies was done as part of her final evaluation. About four hours afterward, she complained of abdominal discomfort and a sensation of fullness in her neck. Roentgenograms and fluoroscopy disclosed a pneumoperitoneum, pneumomediastinum, and subcutaneous emphysema of the neck (fig. 2). Surgical consultation for the first time was requested three hours after the symptoms developed, and seven hours after the barium enema.

Examination: (July 30, 1954). The patient was a well-nourished, small white woman,

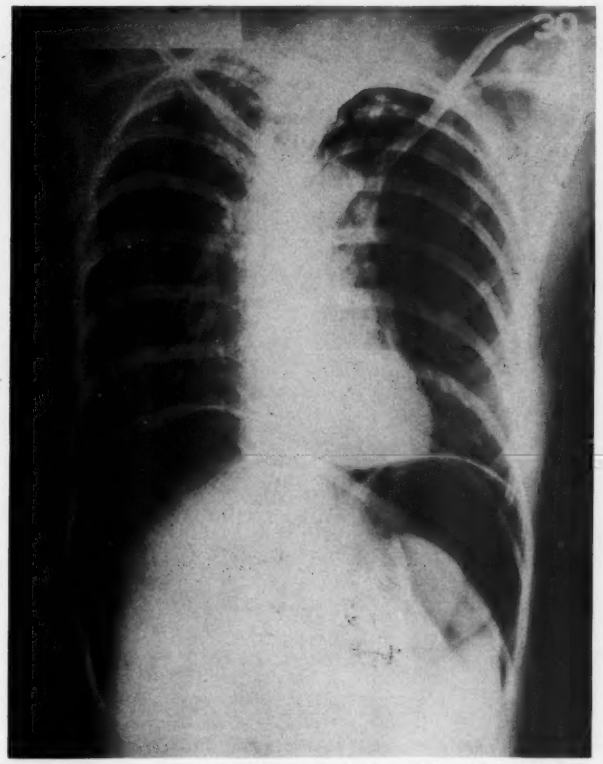

FIG. 2. Upright roentgenogram of abdomen and chest, July 30, 1954, showing pneumoperitoneum following air contrast studies.

lying in bed, conscious, appearing chronically ill but not in acute distress. Her height was 59 inches, and weight was 79 pounds. Her temperature was 96.8 F, pulse 120, respirations 28, and blood pressure 128/80. The positive physical findings were: palpable subcutaneous emphysema of the cervical and supraclavicular regions, distended, resonant abdomen with audible but diminished peristalsis, no particular pain or tenderness on palpation but some slight rigidity in the upper quadrants and epigastrium, a positive Hammond's sign over the precordium, a shortened right lower extremity with ankylosis of the hip, and slight residuals of a spastic hemiplegia on the right.

Laboratory: (July 30, 1954). Urinalysis was normal except for 3 plus albumin and 6 to 10 white blood cells and numerous red blood cells in each high power field. White blood cell

FIG. 3. Photograph of surgical specimen (terminal ileum and right half of colon), showing marked stricture of ileocecal region and involvement of ascending colon by the pathologic process.

count was 15,750 per cu. mm. with 77 per cent neutrophils, 3 per cent nonsegmented forms, 18 per cent lymphocytes, and 2 per cent monocytes. The hemoglobin was 13.4 Gms. and the hematocrit was 45 per cent.

Operation: Emergency laparotomy was done approximately nine hours after the barium enema. When the peritoneum was incised, considerable nonfoul gas under pressure escaped from the peritoneal cavity. The omentum was emphysematous, as was the mesentery. A small pinpoint sized perforation was found on the anterior surface of the ascending colon in an area of subserosal hemorrhage just below the hepatic flexure. The terminal ileum, ileocecal valve, and cecum were involved in a chronic fibrotic inflammatory process which had nearly occluded the lumen of the intestine, and the terminal ileum proximal to this was dilated and hypertrophied, being nearly as large in diameter as the colon. No other areas of obvious enteritis, peritonitis, obstruction, or perforation could be found. There was no fluid or free barium in the peritoneal cavity. A right hemicolectomy with primary end to end transverse ileocolostomy was done and the patient recovered uneventfully.

Pathology: The tissues removed showed no evidence of tuberculosis, either past or present, but did show organisms characteristic, but not absolutely diagnostic, of *E. histolytica* (fig. 3). No stool examinations had been done previous to surgery, but numerous stool examinations after surgery failed to show either the trophozooites or cysts of *E. histolytica*. Proctoscopy on Aug. 23, 1954, showed many pinpoint abscesses and small ulcerations of the rectal mucosa, considered to be diagnostic of amebiasis, and examinations of the smears of these lesions showed organisms suggestive of *E. histolytica*.

Follow-up: The patient was returned to the Medical Department on her seventeenth postoperative day. She was treated for amebiasis with chloroquine and fumadillin but tolerated these drugs poorly, and the treatment was changed to carbarsone and atabrine, which she tolerated well. She was discharged from the hospital on Sept. 9, 1954, to be followed and treated for both amebiasis and pulmonary tuberculosis as an out-patient. When last heard from on Dec. 9, 1954, she was not only doing very well but also had become pregnant since leaving the hospital.

SUMMARY

A discussion of perforation of the colon by barium enema with and without air contrast studies, with a review of some of the other causes of perforation of the colon, including some of the experimental work upon this subject and its relation to the topic under discussion, has been presented. The diagnosis, treatment, and mortality rate of this accident has been discussed briefly. A case report of a patient who, following a barium enema with air contrast studies, had a perforation of an amebic lesion of the ascending colon, which pre-operatively was thought to be tuberculous entero-colitis, has been presented.

CONCLUSIONS

Perforation of the colon during or following barium enema probably is a more common incident than the reports in the medical literature indicate.

Perforation of the colon, whatever the cause, is a catastrophe which requires prompt accurate diagnosis and immediate surgery; the mortality rate resulting from this accident is directly proportional to the time elapsing between its occurrence and operation.

Diseased colons appear to be more easily perforated than normal colons, and, because barium studies of the colon usually are conducted in colons diseased or suspected of being diseased, due care should be exercised in doing this procedure.

The roentgenologist should be fully informed of any facts thought to render the colon more liable to perforation.

Amebiasis must not be overlooked or forgotten in dealing with colonic lesions, and, in that amebiasis results in spontaneous perforation of the colon at times, it seems reasonable to believe that the danger of perforation is increased in making barium studies or other examinations of colons diseased with amebiasis.

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MASSIVE HEMORRHAGE FROM THE EXTRALUMINAL ULCER BED AFTER PARTIAL GASTRIC RESECTION

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Gastrointestinal bleeding in the early postoperative period after partial gastric resection for peptic ulcer may come from one of several sources: the fresh suture line, an early acute marginal ulceration, an unidentified and unremoved lesion (originally mistaken for a bleeding ulcer which has continued bleeding after operation) or the ulcer itself. It is common practice to remove the ulcer if feasible during a partial gastrectomy, and to close the duodenum distal to the ulcer, thereby leaving the ulcer bed in the contiguous tissues.

One of the advantages ascribed to partial gastric resection for massive hemorrhage from duodenal ulcer is the opportunity in most instances to isolate the ulcer bed and in this way, to exclude it from contact with digestive juices. The following 2 case reports demonstrate that massive hemorrhage from such an extraluminal ulcer bed must be considered in the differential diagnosis of postgastrectomy hemorrhage.

CASE REPORTS

Case 1. J. W. C. This 58 year old white man was admitted to the hospital on Nov. 26, 1950 because of ulcer symptoms of three months duration, melena for one month and syncope the day before admission. He also gave a history of alcoholism, chronic bronchitis, arthritis and hypertension. He had had a hemorrhoidectomy for rectal bleeding five months previously.

Physical examination showed an unkempt, poorly nourished white man with a pulse of 108 and blood pressure of 105/65. Coarse rales were heard at the bases of both lungs. No stigmas of cirrhosis were present except for scant chest hair, several spider angiomas and moderate hemorrhoids. Tarry stool was observed on the examining finger.

Laboratory findings: Upon admission the patient's red blood cell count was 3.3 million per cu. mm. The hemoglobin was 67 per cent and the white blood cell count was 19,800 per cu. mm. with 72 per cent polymorphonuclear leukocytes. Bleeding and clotting times were normal. Prothrombin activity was normal. On the night of admission, he was given 1500 cc. of blood and received an additional 1500 cc. during the next 24 hours at which time bleeding appeared to have stopped. A gastrointestinal roentgenographic series, which was done without palpation, showed a scarred duodenum, a deep ulcer crater, and a large paraesophageal hernia. Esophageal varices were not seen, but the Valsalva maneuver was not done. Shortly thereafter the patient suddenly passed 2000 cc. of bright red clotted blood by rectum.

After receiving 1500 cc. of blood, the blood pressure was restored to 125/70 and his pulse to 84. Upon immediate exploration a deep ulcer crater was found to be penetrating into the gallbladder wall and into the head of the pancreas. The pancreaticoduodenal artery was found to be actively bleeding in the ulcer base. Hemostasis was maintained by pressure while the duodenum was dissected beyond the ulcer and a satisfactory closure was obtained

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in uninvolved duodenum. Bleeding from the ulcer itself was controlled with mattress sutures of cotton. Partial gastric resection was completed and a rubber tissue drain was placed in the right upper quadrant of the abdomen in a position to avoid contact with the duodenal suture line. Brisk bleeding occurred during induction of anesthesia and exposure of the duodenum. An additional 3500 cc. of blood was required during the operation and in the next 24 hours before the patient's blood pressure was stabilized.

On the fourth postoperative day he developed left upper quadrant tenderness and gradual distension. The serum amylase was normal. The distension improved with nasogastric suction, but edema of the lower extremities supervened. On the nineteenth postoperative day he began to vomit blood and passed bloody stools per rectum. A slight icteric tint appeared in the scleras. It was believed that he was bleeding from esophageal varices; the risk of bleeding from the old ulcer bed was not recognized. An esophageal tamponade was established but the patient died on the twenty-second postoperative day. At necropsy a demarcated abscess containing 100 cc. of purulent exudate was found in the left upper quadrant under the left diaphragm. The duodenum was distended with clotted blood. A clean sinus tract 0.6 cm. in diameter and 1.3 cm. deep was found to extend through the otherwise well-healed and intact suture line. In its base was an open artery measuring 0.3 cm. in diameter. Microscopically this sinus tract appeared as a continuous pathway between the duodenal lumen and the surgically isolated ulcer bed. The mucosa of the duodenum had partially sloughed; suture granulomas containing minute abscesses where in the duodenal wall.

Originating from intact mucosa, a single cell layer of epithelium extended across the exposed duodenal submucosa, mucularis and serosa and then abutted on the scarred ulcer bed. At this point a small abscess, which incorporated many foreign body giant cells created a shallow excavation. Beyond this abscess an organizing fibrin membrane led into and was continuous with the thrombotic lining of a large open vessel.

Case 2. P. L. G. This 66 year old white man was admitted to the hospital on Oct. 11, 1953 because of hematemesis. His history was consistent with a duodenal ulcer of 30 years' duration. Physical examination showed a confused elderly man with a pulse rate of 80 and blood pressure of 144/90. There was slight epigastric tenderness.

Laboratory findings: Upon admission the patient's hemoglobin level was 8.4 grams per cent at which time he was given 500 cc. of blood. On the following day an upper gastrointestinal roentgenographic series done without palpation showed a large ulcer in the prepyloric and duodenal areas. On the same day he had further hematemesis and went into shock. A rapid transfusion restored his blood pressure to normal. The abdomen was explored within an hour of the bleeding episode and a large ulcer was encountered that involved the right lobe of the liver and the head of the pancreas, and extended proximal to the pylorus. A perforation into the gastrohepatic omentum was found and brisk bleeding was present from a major artery in the ulcer crater. The duodenum was mobilized beyond the ulcer and a partial gastric resection completed. Bleeding from the ulcer bed was controlled with sutures of silk. A rubber tissue drain was placed in the right flank, in such a position as to avoid contact with the suture lines.

The patient's postoperative course was complicated by atelectasis and a wound hematoma. Throughout the postoperative period he had a low grade fever and moderate epigastric discomfort. On the eighteenth postoperative day a massive hematemesis occurred. Because of his critical condition one attempted operation had to be discontinued after temporary control of the bleeding by sutures in the pancreas. Bleeding recurred almost immediately through the nasogastric tube and the drain site. Eight transfusions were given and the patient was re-explored. Bleeding was again visualized in the region of the necrotic pancreas, and after more complete mobilization of the duodenum, the bleeding point was again controlled with mattress sutures of silk. An incision was also made in the anterior duodenum and a defect could be demonstrated in the duodenal wall posterior to the suture line and in communication with the region of the actively bleeding vessel.

The round ligament was mobilized and sutured into the posterior duodenal defect. A

catheter was left through the duodenal wall for decompression. He improved considerably during the operation, during which time five further transfusions were given.

No further bleeding occurred postoperatively, but he remained in a critical condition with a rapid pulse and evidence of extensive bronchopneumonia. He became progressively lethargic and developed peripheral edema. Venous pressure and circulation time were normal. Aberrations in the electrolyte pattern were corrected, but he died on the twenty-third postoperative day.

Necropsy showed extensive bronchopneumonia and marked cardiac dilatation, fibrosis and atherosclerosis. There was no further evidence of duodenal leakage or hemorrhage.

COMMENT

The actual sequence of events that allowed extraduodenal hemorrhage to gain a pathway back into the duodenum cannot be reconstructed. It is conceivable that leakage of pancreatic enzymes from the ulcer bed in the pancreas caused necrosis of the adjacent duodenum and necrosis of the ligated vessel. The role of the drain in adversely affecting the healing of the duodenum must be suspected, but both drains were placed to avoid the suture lines and in the second case the duodenal defect was separate from the suture line. The possibility that necrosis of the duodenal wall was caused by deep ligatures that were placed to control the hemorrhage from the pancreaticoduodenal artery also must be considered. It would seem advisable to ligate the arteries feeding the ulcer area at a distance from the ulcer bed, if possible. In neither instance was there extensive dissection and devascularization of the duodenal tissues. In neither instance was there a frank gross dehiscence of the duodenal suture line but presumably disruption must have occurred in a microscopic form in the first case. The mechanisms producing this complication are not clear.

CONCLUSIONS

Although few suggestions can be made as to a means of avoiding it, these cases are presented to call attention to the ulcer bed as a possible site of postoperative intraintestinal hemorrhage even when the ulcer is believed to have been excluded from access to the duodenal lumen during the performance of emergency gastric resection for bleeding. The presence of an inflammatory reaction in the region of the duodenal stump, even without frank disruption, should lend weight to this possibility.

MEGAILEUM WITH PARTIAL STRANGULATION*

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Megacolon (Hirschsprung's disease) is an obstruction of the colon with localized dilatation and hypertrophy without gross change in the lumen. Similar lesions of the esophagus (megaesophagus)[18] and of the duodenum (megaduodenum)[1, 7] have been reported in the literature but none involving the jejunum or ileum. This paper is written to record such a case.

CASE REPORT

C. B. S., a 10 year old boy, entered the hospital with a diagnosis of *"acute abdomen"*. He gave a history of chronic constipation, indigestion, peculiar appetite, abdominal pain and discomfort since birth. His father stated that at 6 months of age a roentgenogram was made and he was told that the child would need surgical correction for an *"abnormality of the bowel"*. The patient's acute abdominal episodes had been treated previously with water and glycerin enemas which served to give relief after a short time.

The physical examination was negative except for the abdomen which was slightly distended and diffusely tender throughout. There was no localized pain. The temperature was normal, the white blood cell count was 12,300 per cu. mm. Fluids and sedation produced no improvement in the first 12 hours. At the end of this time the white cell count had risen to 24,200 per cu. mm. A barium enema showed a normal colon with no evidence of obstruction or constriction. The cecum lay in the midline and just below the transverse colon. There was much distention of the stomach. Immediate operation was advised. On opening the peritoneal cavity the following operative note was recorded: "There was a congenital malrotation of the mesentery with a large Meckel's diverticulum and an anomalous condition of the transverse colon whereby it passed through instead of around the superior portion of the mesentery. There was a volvulus of the terminal ileum and ascending portion of the colon with thrombosis of the mesenteric vessels and marked enlargement of the lymph nodes at the base of the mesentery. The small bowel above Meckel's diverticulum was markedly dilated and the cecum was dilated and filled with fecal material. There was an obstruction of the colon at the point where it perforated the mesentery in the midportion of the transverse colon. The Meckel's diverticulum was approximately 20 cm. in diameter and filled with air and some fluid. A good deal of difficulty was encountered in identifying the malrotation as it existed in this patient. The Meckel's diverticulum which was found behind the stomach finally was delivered, the small bowel sectioned at a point approximately 30 cm. proximal to Meckel's diverticulum and the transverse colon cut at the point where it passed through the mesentery of the small bowel. Resection of a large part of the ileum, ascending colon, cecum and first part of the transverse colon was completed. The small bowel was anastomosed end to side with the transverse colon. The mesenteric defect was closed with interrupted sutures of chromic catgut and the abdominal incision was closed. The immediate postoperative condition was fair."

The postoperative course was satisfactory. On the day following the operation the tem-

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perature was 102 F. then returned to normal the next day. On the following day it arose again to 100 F. and there was some vomiting which was relieved by gastric suction and the general condition improved steadily from that time on. He was placed on a regular diet six days after operation at which time a barium enema showed the enterocolostomy with a normally functioning anastomosis. He was ambulated on the eighth day without ill effects at which time the wound was well healed and he was dismissed. He said that he felt better than he ever felt in his life.

The pathologist's report: The material received had been preserved in formalin and was composed of a cystic intestinal mass, embedded in the center of a large segment of the small intestine and attached mesentery. The small intestine included a widely rounded dilated segment of the ileum 13 cm. in diameter located 43 cm. from the ileocecal valve and 47 cm. from the proximal end of the resected intestine. This dilated segment of the small intestine is described in the surgeon's note as a Meckel's diverticulum. This saccular dilatation had two wide openings 8 cm. apart. The lower opening measured 18 mm. in diameter and opened into the lower end of the ileum which had gradually increased in diameter to 30 mm. where it entered the ileocecal valve. The wall of this part of the ileum, especially the mucosa, gradually increased in thickness to the cecum. The lymphoid tissue also was increasingly prominent, lymphoid follicles being recognized in the tips of some of the hyperplastic epithelial folds.

The other opening (upper) in the saccular dilatation was 27 mm. in diameter and was situated 8 cm. above the lower aperture and opened directly into the upper segment of the ileum. The mesentery was attached to the outer surface of this localized dilatation that connected the two openings. The wall of this saccular mass was thickened, especially the musculature, measuring 2 to 4 mm. in thickness and its inner surface was lined by hyperplastic velvety mucosa thrown into irregular partly flattened folds continuous with the mucosa of the adjacent ileum. Its inner surface showed a few scattered small hemorrhagic superficial ulcers, the largest of which measured 7 by 3 mm. in size.

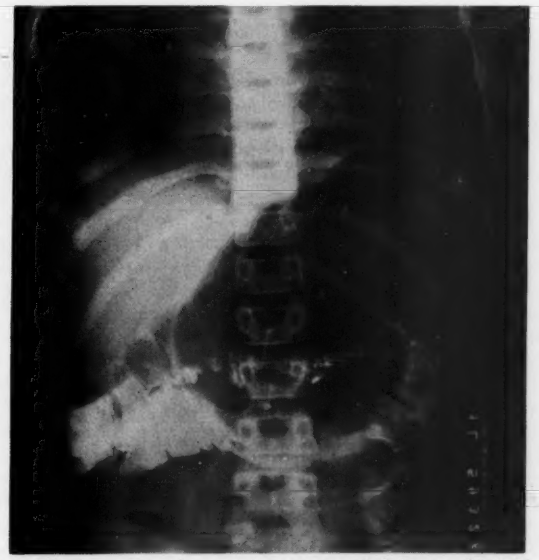

FIG. 1. Photograph of preoperative roentgenogram of abdomen. Note the large rounded cystic mass (megaileum) in the upper left quadrant.

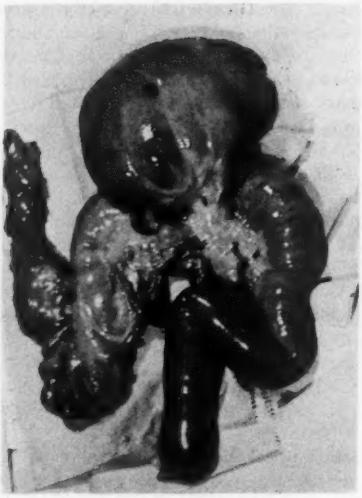

FIG. 2

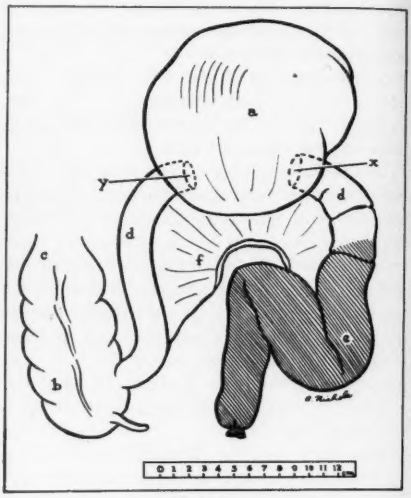

FIG. 3

FIG. 2. Photograph of specimen taken immediately after operation. Note the cystic mass (megaileum) arising from the small intestine. Note also the cecum and ascending colon on the left and the discolored strangulated proximal end of the small bowel on the lower right.

FIG. 3. Diagram of the specimen in figure 2 showing the relation of the saccular dilatation (a) of a segment of the ileum (megaileum) to the cecum (b) and to the strangulated proximal end (e) of the small intestine. Note the location of the two openings (x and y) of the sac into the ileum, their size and their position apparently behind the saccular dilatation (dotted lines). Note also the mesentery below and attached to the dilated ileum.

The segment of the small intestine above the sac was 47 cm. in length and included the upper end of the ileum. The first 3 cm. above the dilatation measured 27 to 30 mm. in diameter and then the lumen dilated rapidly to 45 mm. towards its proximal end. The proximal one-half of this segment, twisted on itself, was thin-walled, distended and discolored, with obliteration of most of the epithelial folds. It appeared to be strangulated. The lower one-half of this segment showed less discoloration and a much thicker wall as it approached the saccular dilatation. The discolored thin-walled upper part probably was the upper end of the ileum.

The vessels in the attached mesentery were engorged and often thrombosed, especially those going to the discolored segment. The mesenteric lymph nodes were enlarged measuring 1 to 3 cm. in diameter and appeared edematous and hemorrhagic on section. The serous surface over both the intestines and mesentery was dull and lusterless.

The proximal segment of the colon included the ileocecal valve, the cecum, appendix, the ascending colon and a small segment of the transverse colon. The lower end of the ileum entered the ileocecal valve in the normal way. The cecum and colon was 14 cm. in length and 22 to 47 mm. in diameter, the small end being the small part of the transverse colon. The appendix was 9.5 cm. in length and appeared normal. Lymphoid tissue was prominent in both the lower end of the ileum and in the cecum and ascending colon.

Microscopic Findings: There was marked hyperemia throughout the entire intestinal tract and mesentery with focal hemorrhages, thromboses and collections of polymorphonuclear leucocytes especially towards the discolored thin-walled proximal end. Focal collections of polymorphonuclear cells were scattered throughout the intestinal wall frequently associated with a perivascular reaction in the submucosa. There was no fibrin on the surface of the peritoneum but the vessels were congested and often surrounded by both

polymorphonuclear and mononuclear cells and some contained thrombi. The same mild inflammatory reaction was seen throughout the wall of the saccular dilatation. In the mucosa a few superficial hemorrhages and erosions also were seen. The lymphoid tissue and follicles were very prominent, especially in the distal end of the ileum. The muscular wall of the saccular dilatation was definitely hypertrophied. The mesentery and lymph nodes showed marked hyperemia with some focal hemorrhage, edema and scattered clumps of polymorphonuclear cells.

Numerous sections were made to study the myenteric plexus and Meissner's plexus in all parts of this material. Especially numerous were the blocks removed from the saccular dilatation, at the lower opening and the segment of the ileum from the dilated part to the cecum. In fact, serial sections extending from the lower opening of the saccular dilatation to the cecum were thoroughly studied. Occasional blocks showed a few degenerated ganglion cells or some diminution in number but this also was observed in the wall of the saccular dilatation and in the ileum above the saccular dilatation. In no place was there consistent evidence of degenerated or absent ganglion cells either in the myenteric (Auerbach's) plexus or in Meissner's plexus. In fact the nerve cells and plexi appear unusually well developed in the lower end of the ileum.

Pathologic Diagnosis: Congenital focal idiopathic megaileum, early acute diffuse ileocolitis and mesenteric cellulitis with focal thromboses. Strangulation of upper ileum. Acute hemorrhagic lymphadenitis.

DISCUSSION

The clinical, gross and microscopic changes in this patient are similar to those seen in sigmoid megacolon,[3, 4, 6, 8, 9, 11, 12, 13, 14, 15, 16] megaesophagus[5] and megaduodenum. There is the same history of a gastrointestinal anomaly dating from birth, and a localized dilatation and hypertrophy of the intestinal wall with obstruction but no organic occlusion of the lumen. On the other hand there is no evidence of disturbance of the myenteric plexus as is described by Whitehouse and Kernohan[19] in cases of congenital megacolon. Not all cases of congenital megacolon[2, 10] show absence or degeneration in the ganglion cells in the constricted part of the colon below the dilated colon. Some authors[9] deny the existence of a so-called idiopathic megacolon while others classify congenital megacolon into several types including one in which there is no satisfactory explanation of its cause.

Apparently, in this child, whom we have just described, a volvulus developed in the small bowel above the sac, producing strangulation with subsequent ileocolitis and symptoms of an acute abdominal condition. Complete recovery followed resection of the strangulated intestine and the congenital anomaly. Since there is no abnormality in the myenteric nerve plexus and no satisfactory explanation for the development of this congenital anomaly we have regarded it as a congenital idiopathic megaileum.

Although a statement was made at the beginning of this paper that we had not found a megaileum recorded in the literature, there are 2 cases reported which we would regard as possible examples of megaileum—one described in 1935 by Perrot and Danon and mentioned in the paper by Whitehouse and Kernohan[19] and by Tifflin[17] of an infant 15 days old with an intestinal obstruction in the lower ileum showing a dilatation and hypertrophy above a constriction of the ileum without obstruction to the lumen but with degenerated or absent ganglion

cells in the constricted part of the ileum and in the ascending colon. The other is the ninth case reported in 1951 by Zuelzer and Wilson[20] who described a Negro infant 5 days old with a dilated and hypertrophied ileum and a constricted ileum beginning 22 cm. above the ileocecal valve. Ganglion cells were absent in the constricted part and in the entire colon.

SUMMARY

A case of congenital megaileum is reported with successful resection. It is to be classed with megaesophagus and megaduodenum and sigmoid megacolon since it represents a congenital dilated and hypertrophied segment of the gastrointestinal tract with no organic obstruction in the lumen of the involved intestine.

Since the ganglion cells are intact in the myenteric plexus it is a unique example of an idiopathic congenital megaileum.

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SURGICAL TECHNIC

THE SURGICAL MANAGEMENT OF SACRAL AND PRESACRAL TUMORS*

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Sacral and presacral tumors as a whole are relatively rare and the diagnosis often is difficult. Ross[5] found only 7 in 440,255 admissions to the King's County Hospital over a period of 7 years. Others have reported a similar incidence. In the Sanders Clinic, the chondrosarcoma reported herein is the first tumor found in this region in 54,020 patients observed over a period of almost 32 years.

That chondrosarcomas are one of the more unusual types of tumors occupying the presacral space, is indicated by the fact that of 161 tumors in this area reported by Mayo and associates,[3] only 4 were of the cartilaginous type. The majority are congenital (chordoma, dermoid cyst, and teratoma) and neurogenic (ependymoma, neurofibroma, and neurilemmoma). The major portion of the neurogenic group are ependymomas.

In the past, the results of treatment of these tumors have been rather unsatisfactory. Usually, the tumors were partially removed by local resection or curettage and roentgen therapy was given postoperative. The transperitoneal anterior approach, employed by some surgeons, permits only partial resection. Many of the unsatisfactory results are due to the fact that removal of sacral and presacral tumors involves the fields of general, orthopedic, and neurologic surgery. Therefore, the management outlined by MacCarty and associates,[2] whereby a specialist in each of these fields cooperates in the surgical procedure was followed in the case reported below. Our purpose in presenting the case is to emphasize the advantage of this method.

CASE REPORT

The patient, a man aged 33, was seen at the Sanders Clinic on July 22, 1953. He complained of a mass on the buttock and constipation. Ten years earlier he had noticed a small growth on the lower right buttock in the sacroiliac region. At that time, he had an aching sensation in the rectum, although this soon disappeared. He had no further trouble until eight months prior to our observation, when the tumor began to enlarge and he became constipated. The difficulty had increased until laxatives and enemas were totally ineffective. A partial loss of sensation in the perianal region also was reported. He had no other gastrointestinal disturbances, although his appetite was only fair and he had lost 12 pounds in weight, during the eight months prior to admission.

On examination, a large mass was found protruding above the surrounding area just to the right of the midline in the region of the sacrum. The mass was firm and fixed to the sacrum, but was not tender. On digital examination of the rectum, the tumor was found to fill the entire pelvis; at about 4 cm. from the anus, practically complete obstruction was

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encountered. The tumor seemed to be 15 to 20 cm. in diameter. Physical examination otherwise was negative.

Blood studies showed no anemia or other abnormality. Roentgenograms showed a round, smooth, mottled, calcified tumor 15 to 20 cm. in diameter projecting forward from the sacrum and displacing the rectum to the left and far anteriorly (figs. 1 and 2). The remainder of the colon, the kidneys, and urinary tract were normal. There was no evidence of metastases in the lungs or ribs. The tumor was believed to be an osteochondroma, or low grade chondrosarcoma.

The colon was prepared with Aureomycin for three days, and the tumor, with a portion of the sacrum, was removed on July 29, 1953. Prior to operation, the anterior displacement of the rectum so compressed the bladder and ureters that a urologist was unable to insert ureteral catheters.

Following anesthetization, the patient was placed in the Kraske position. A midline incision was made from the fifth lumbar vertebra to the tip of the coccyx. Skin flaps were elevated laterally on both sides. The gluteus muscles were detached from the sacrum and sacroiliac areas and reflected laterally; the coccyx was resected and the coccygeal muscles, sacrotuberous and sacrospinous ligaments were divided.

A hand was inserted into the presacral space, and it was found that the tumor was fairly well encapsulated anteriorly and was at least 20 cm. in diameter. It extended approximately 8 cm. through the gluteus muscles and tissues posteriorly and 10 to 12 cm. anteriorly into the pelvis, occupying practically the entire pelvis and compressing the bladder and rectum. An attempt was made to identify the second, third, and fourth sacral

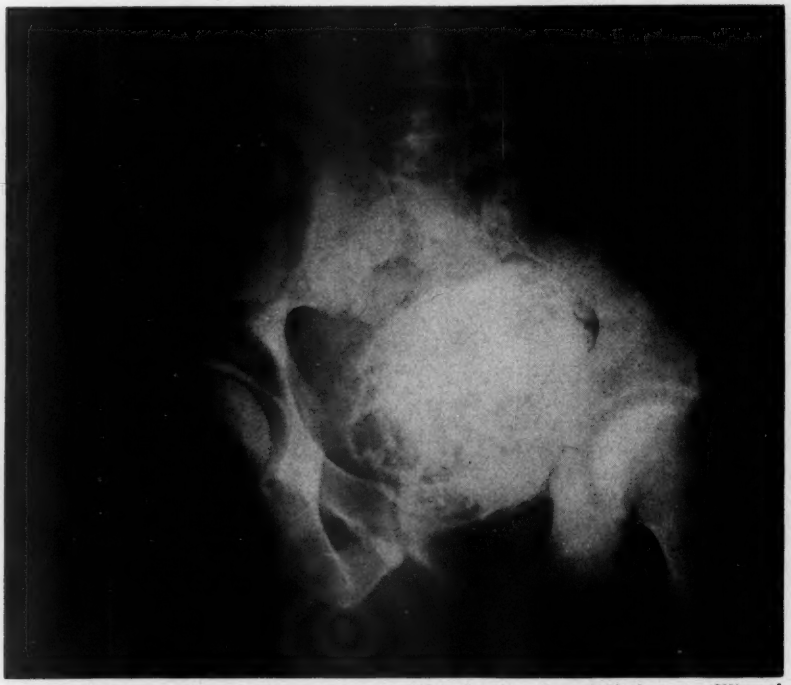

FIG. 1. Anteroposterior roentgenogram of pelvis, showing the calcified tumor filling the entire pelvic space.

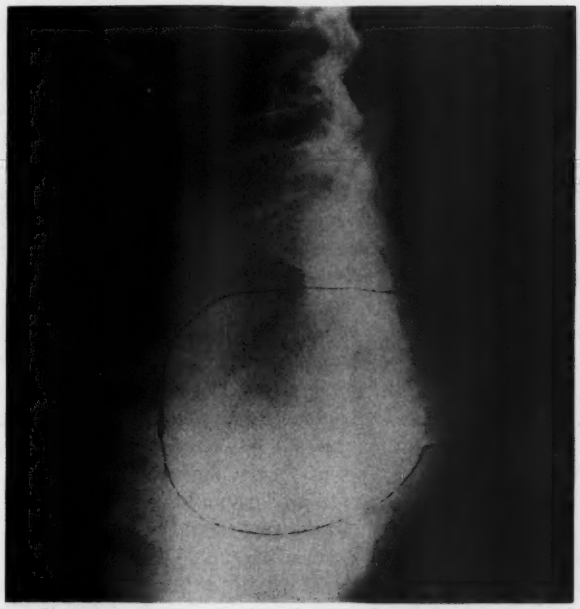

FIG. 2. Lateral roentgenogram, showing the anterior and posterior projection of the sacral tumor.

nerve roots. The tumor extended so high, however, that it had completely enveloped the third and fourth roots, and a special effort to isolate the third root, in order to preserve bladder function, was unsuccessful. In view of the nature of the growth, its complete excision was believed advisable, despite the probable damage to urinary function.

The sacrum was divided across at the junction of the second and third sacral vertebrae. Because of its large size, the tumor could not be removed intact through the space between the sciatic notches; therefore, it was removed in segments, apparently in its entirety, together with the lower sacrum. After ligation of the bleeding vessels, the wound was copiously irrigated. The rectum was inspected and found to be uninjured. Before closure of the wound, two Penrose drains were inserted into the space previously occupied by the tumor, and one drain was placed in the subcutaneous tissue. The patient received a continuous transfusion during the operation, and his condition was good at its conclusion.

Following the operation, it became apparent that the anal sphincter and bladder were partially paralyzed. Since the patient had only partial incontinence of the bowel, the evacuation was controlled by enemas. A temporary suprapubic cystostomy was done. The next day, an extravasation of urine into the abdominal wall around the cystostomy opening was observed. This necessitated a second operation for drainage of the space of Retzius and prolonged the patient's hospital stay an additional 10 days.

Upon dismissal from the hospital five weeks postoperative, he still had a slight drainage from both the posterior and anterior wounds, and was referred to his local physician for observation. Six weeks later he returned to the clinic. At that time, the posterior wound had healed completely, and his bowel function was normal. Since he was voiding through the urethra, the suprapubic catheter was removed. During this procedure, an encrustation was dislodged from the catheter into the bladder. This encrustation was removed transurethrally without difficulty. At his last observation, 20 months postoperative, the bowel and bladder functions were satisfactory, and his control over both was excellent. Further

roentgenograms of the pelvis and chest showed no evidence of local recurrence or metastasis of the tumor. The patient was driving a bulldozer and was not at all disabled.

DISCUSSION

Chondrosarcoma of the sacrum does not differ particularly from chondrosarcoma elsewhere within the body, although, in common with all sacral and presacral tumors, its surgical excision presents problems of preservation of bladder and bowel function, as well as adequate exposure and complete excision. Tumors in these areas are either neoplastic or inflammatory. The recent reports by Jackman and co-authors[1] and by Mayo and associates (table I)[3] discuss fully the various types of lesions.

These tumors seldom produce symptoms until they attain a fairly large size. The symptoms are chiefly those of pressure upon the rectum or bladder, or of destruction or pressure upon the nerve supply to these organs. Progressive bowel and bladder dysfunction, low backache, or pain and paresthesias in the perianal region are the most common complaints. In advanced cases, partial paralysis with paresthesias in the legs or feet may be experienced.

Physical examination rarely shows any palpable or visible tumor externally. Digital examination of the rectum, however, should reveal the mass and permit one to determine its size, contour, consistency and mobility. Rectal and pelvic examination in the female should enable one to differentiate such tumors from intrapelvic genital masses. Proctoscopic examination should be made to rule out fistulas, abscesses and the possibility of invasion of the rectum, or a primary

TABLE I

*Differential diagnosis of presacral tumors in 161 cases**

Tumors	Per Cent
Congenital	
Chordoma	32
Dermoid cyst	18
Teratoma	12
Inflammatory	11
Neurogenic	
Ependymoma	7
Neurofibroma	3
Neurilemmoma	1
Osseous	
Giant cell tumor	3
Osteogenic sarcoma	2
Cartilaginous tumor	2
Osteoma	1
Miscellaneous	
Metastatic carcinoma	2
Myeloma	2
Others	3

* Mayo, C. W., and associates.[3]

rectal tumor. Roentgenologic study of the colon and sacrum is necessary to complete the preoperative evaluation.

Preoperative Care. The primary feature of the preoperative care is the preparation and sterilization of the colon. In the case reported, Aureomycin was used, although we now follow the plan suggested by Poth and co-workers,[4] i.e., Neomycin, 1 Gm. with Sulfathaladine, 1.5 Gm., orally every four hours for three days, with carthartics and daily enemas. Transfusions and other supportive treatment may be indicated.

Operation. Following administration of a general anesthetic, the patient is placed in the Kraske position and the field prepared and draped. The drape edge is sutured to the skin just above the anus, sealing off this area of contamination.

A longitudinal midline incision is made from the spine of the first sacral vertebra to the tip of the coccyx, and the fat, fascia, and gluteus maximus muscles are dissected free and widely retracted laterally (fig. 3).

If uninvolved by the tumor, the coccyx is disarticulated and removed. The hand is insinuated into the retrorectal space, freeing the tumor from the rectum

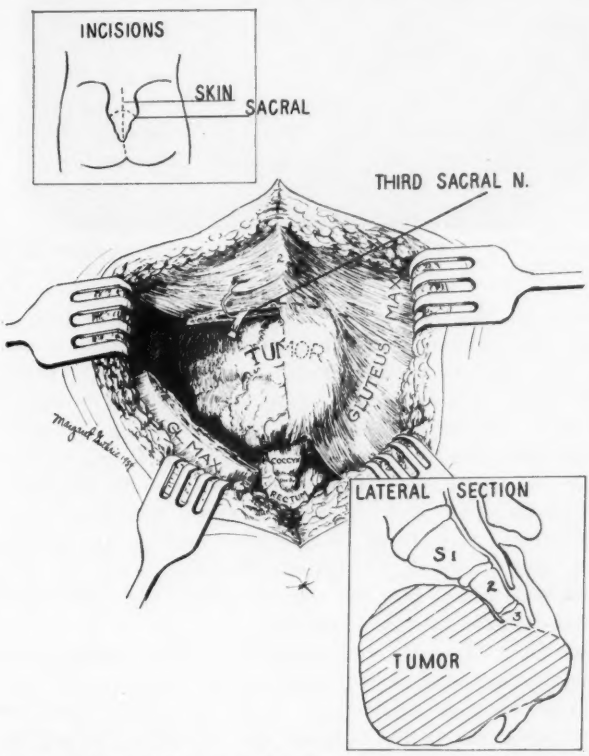

FIG. 3. Upper inset showing location of incision. Lower inset showing a lateral view of the tumor. The main drawing showing reflection of muscles and partial resection of the sacrum.

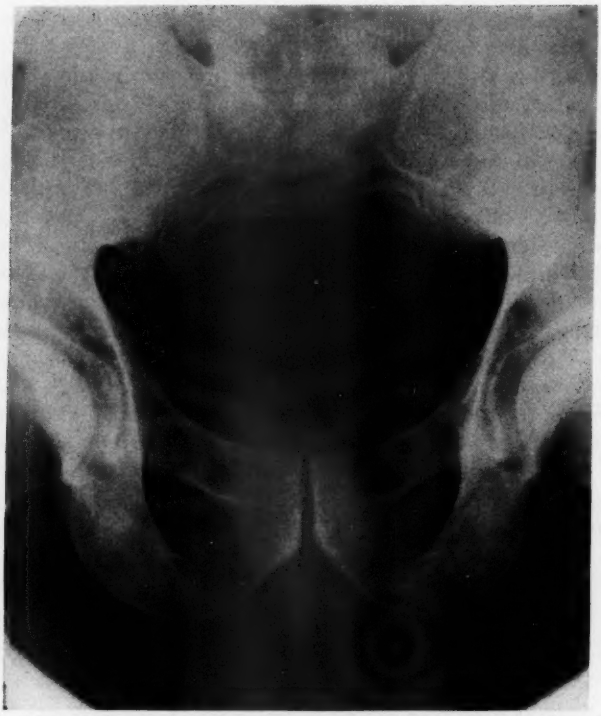

FIG. 4. Posteroanterior roentgenogram one year postoperative showing short sacral stump with no evidence of recurrence.

by blunt dissection. This maneuver is best done by the general surgeon. Next, the orthopedic surgeon should identify and divide the sacrotuberous and sacrospinous ligaments and the pyriformis and coccygeal muscles. Thereafter, the neurosurgeon and orthopedist should combine their efforts in identifying and dividing the lower two sacral nerve roots, and identifying and preserving the pudendal nerves and the second and third roots. The sacral arch then is cut across between the second and third, or third and fourth segments, the filum terminale is divided, and the tumor and involved portion of the sacrum are removed. Following this, the rectum should be carefully inspected, and if any injury or perforation is found, it should be repaired. The operation is completed by inserting two Penrose drains into the resultant dead space and reapposing the gluteus maximus muscle, the fascia and skin.

Postoperative Care. The catheter is removed from the bladder after three or four days. If any evidence of bladder denervation is observed, the catheter is reinserted and tidal drainage is carried out, or, if necessary, a cystostomy is done. A neurologic examination of the involved area should be made after the postoperative condition of the patient is stabilized. This will demonstrate the extent of nerve damage and provide a basis for prognosis and a plan for rehabilitation.

SUMMARY

The combined efforts of the general surgeon, neurosurgeon, and orthopedist offer possibilities for improvement of the results of treatment of certain sacral and presacral tumors.

Often in the past these tumors have been inadequately removed. In other cases, when removal was complete, bladder and rectal paralysis remained. Paralysis consequent upon radical resection occasionally is justified in order to remove a malignant or potentially malignant tumor in its entirety; however, complete removal, without resultant disability, often can be safely effected by an operative team, as described.

Sacral and presacral tumors should be kept in mind as a possibility when a patient complains of low backache, bowel symptoms, or similar disturbances.

The case reported illustrates a method for handling some of the problems presented by tumors in the presacral region. The follow-up period is too short to permit any conclusions regarding cure.

Obviously, all tumors of the sacrum and presacral space do not require radical resection, as described. Benign tumors, such as dermoid cysts, inflammatory tumors, and the like, can be satisfactorily managed without resection of portions of the sacrum.

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2. MacCarty, C. S., Waugh, J. M., Mayo, C. W., and Coventry, M. B.: Surgical treatment of presacral tumors; combined problem, Proc. Staff Meet. Mayo Clinic, *27:* 73 (Feb. 13) 1952.
3. Mayo, C. W., Baker, G. S., and Smith, L. R.: Presacral tumors; differential diagnosis and report of case, Proc. Staff Meet. Mayo Clinic *28:* 616 (Nov. 4) 1953.
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EDITORIAL

THE FUTURE OF ACTH AND CORTISONE IN SURGERY

The knowledge that operative trauma as well as ACTH produces adrenocortical stimulation with resultant increased secretion of steroid hormones has given rise to much speculation and experimentation. In the first place, is this reaction beneficial to the patient or is it simply an expression of increased catabolism and perhaps harmful? Slight evidence is being presented, and certain ideas have been expressed by various authors, to the effect that the reaction is beneficial to the patient. If it is beneficial there would be fairly valid pharmacologic reasons why ACTH and certain adrenal steroids might improve a patient's condition if he were suffering from certain types of insufficiency.

The stimulation mentioned above results in an increase in secretion of 11-oxysteroids, related hormones designated as 17-hydroxycorticoids and a doubtful increase in the 19-ketosteroids. Recent work has confirmed earlier impressions that the urinary excretion of 17-ketosteroids after trauma is variable, although one group of investigators has obtained a fairly consistent increase by measuring the acidic as well as the neutral fractions.

It has been stated that 80 per cent of the steroid hormones secreted as the result of hypothalamic-pituitary-adrenocortical secretion following operation is hydrocortisone. Although the therapeutic action of hydrocortisone and cortisone essentially is the same, it must be remembered that they are different compounds. Since hydrocortisone is available in a soluble form (free alcohol), and can be given intravenously, its effects are more intense.

In any consideration of the use of ACTH and cortisone or hydrocortisone in surgical patients, the surgeon must constantly bear in mind the deleterious effects of these hormones, which have been summarized elsewhere. One of the most important of these effects is production of a negative nitrogen balance. Important also is retention of sodium, chloride and water; but an increased secretion of potassium, calcium and phosphorus is almost as important.

Although some authors have reported rather definite beneficial effects of ACTH and adrenal corticoids (cortisone and hydrocortisone) in improving operability, other observers have not been impressed with the value of these drugs to the surgeon. The explanation of this inconsistency may lie in the choice of patients and lack of knowledge concerning indications. Unfortunately, there is no reliable test for adrenocortical insufficiency. Perhaps the drop in the eosinophile count (Thorn) is the most reliable index of cortical insufficiency, but rather extensive use of this phenomenon as a test by numerous observers has not indicated the eosinophile count is a truly reliable test.

In spite of the lack of a test, clinical impressions sometimes are quite accurate in determining whether or not the steroid hormones will be of any value in improving operability. For example, we have no evidence that a patient aged 35 to 50 having cholelithiasis, but with normal food intake and nutrition, will receive any clinical benefit from preoperative steroid hormone therapy; in fact,

such patients probably would be harmed by such therapy. On the other hand many elderly patients with anorexia, malnutrition, or fever from chronic infection often are improved markedly with 5 or 6 days' therapy with these drugs. It appears rather obvious that the clinician seeing only private patients with normal nutrition will see very few who could be benefited by steroid therapy. On the other hand the clinician working in a charity clinic, where elderly, undernourished patients are encountered with such high frequency, might see many who would be benefited. It is not fully appreciated that aged people lose their appetite even with mild disease, and thus are quite susceptible to malnutrition. It must be remembered that such patients tend to gravitate to charity clinics because their earning power is low or absent.

From the above remarks I am not implying that every patient with nutritional depletion has adrenocortical insufficiency and will benefit from steroid therapy. However, I am convinced that in at least a few of these patients, operability is improved markedly by steroid therapy. On some occasions a true adrenal insufficiency may be present, perhaps from some other cause, and steroid therapy will for this reason be very helpful. On other occasions it is perhaps helpful because it produces a euphoric, antipyretic effect, and increases appetite as well as the desire to be ambulatory. Since the hormones under discussion tend to result in a negative nitrogen balance, a patient given these agents must be placed on an extremely high caloric diet (3500 to 5000 calories) and be given a relatively low fluid intake to minimize water retention. In our clinic we have had more experience with ACTH than with cortisone or hydrocortisone and have given it in doses of 100 mg. per day for 5 or 6 days preoperatively. Either one of the three drugs should be continued in decreasing doses for 2 to 4 days postoperatively, particularly if cortisone or hydrocortisone has been given; this would minimize the possible ill effects of cortical atrophy after operation.

It is well known that these hormones result in marked temporary improvement in ulcerative colitis. If operation is contemplated in such patients and operability is doubtful, a short course (4 to 6 days) of one of these drugs often will convert inoperability to safe operability.

In certain hematopoietic diseases of the purpura and hemolytic anemia type, steroid therapy usually produces a marked temporary beneficial effect. Under such circumstances, patients who are poor risks may be converted to a state of fair operability if operation appears indicated.

Although it now is well established that ACTH and cortisone are of no benefit (may even be harmful) in shock due to hemorrhage, yet evidence is accumulating to the effect that in certain other types of shock benefit may be derived from these drugs.

The antiallergic effect of these drugs is well known. Although no single clinician will encounter a significant number of patients with drug reaction, or serum sickness, to give these drugs a fair trial, they would appear to be indicated on these occasions. Likewise, they might be very beneficial if given intravenously in the early stages of a transfusion reaction.

From the above remarks, it is obvious that an efficient test to determine the need of steroid therapy, particularly as a preoperative measure would be very useful. Such a test might reveal whether ACTH was indicated for a *priming* action to insure a satisfactory steroid output during surgery, or might reveal the need for specific substitution therapy during and after surgery. Efforts are being made in our laboratory and many others, to devise a relatively simple test to determine when a patient with poor operability can be improved with steroid therapy. It is reasonable to believe that these efforts will be successful, at least to a slight extent.

Accordingly, it is the author's opinion that ACTH, cortisone and hydrocortisone are of benefit in improving operability in certain patients, but in the majority of patients probably would be harmful. Likewise it appears probable that in the not too distant future tests will be found to give us an indication as to when such drugs should be given.

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BOOK REVIEWS

The editors of THE AMERICAN SURGEON *will at all times welcome new books in the field of surgery and will acknowledge their receipt in these pages. The Editors do not, however, agree to review all books that have been submitted without solicitation.*

Pathology for the Surgeon. By WILLIAM BOYD, M.D., Edin., Dipl. Psychiat. Edin., F.R.C.S. Canada, F.R.C.P. Lond., M.R.C.P. Edin., F.R.S. Canada, LL.D. Sask., D.Sc. Man., M.D. Oslo. Lecturer on the Humanities in Medicine, the University of Toronto; Visiting Professor of Pathology, the University of Alabama. Formerly Professor of Pathology, the University of Manitoba, The University of Toronto and the University of British Columbia. Seventh Edition, with 547 illustrations including 10 in Color. W. B. Saunders Company, Philadelphia & London, 1955. Price $12.50.

For thirty years Boyd's Surgical Pathology has occupied a prominent position in the libraries of most students and practitioners of surgery. The title of this the seventh edition, Pathology for the Surgeon, will probably strike a slightly dissonant note to most of us who have become so accustomed to the old familiar title. However, as stated by the author, "an old suit may be patched and repaired so as to make it reasonably presentable, but the time comes when it begins to get dated." Therefore, much of the material has been rewritten rather than revised and the emphasis has been directed toward the needs of the young graduate surgeon wishing to refresh his knowledge of pathology with considerable clinical and practical material interspersed with the basic considerations of pathology. Likewise the term "surgical pathology" has assumed new connotations in recent years with the development of subspecialists in surgical pathology who direct much of their attention to the examination and diagnosis of fresh surgical material, "the pathology of the living." However it is not this aspect of pathology that the author wishes to emphasize but rather the broader picture of lesions and disease processes occurring in those conditions commonly treated by the surgeon with correlation between the changing clinical picture and the pathologic changes found in the tissues; hence, the present title, "Pathology for the Surgeon".

The material is divided into two broad categories, General Surgical Pathology and Special Surgical Pathology. Several new chapters have been added to keep abreast of the rapidly changing field of surgery. Numerous excellent illustrations have been added and the entire volume is well organized for easy readability. Above all the beautiful writing style for which the author is so well known makes study a pleasure. His description of the mucosa of a fresh gallbladder as "tall graceful folds and membranes, gossamer-like in their delicacy, seen floating like seaweeds in a marine pool" makes a thing of beauty of this oft maligned organ. In the section on the lungs: "to reconstruct the development of tuberculosis from an examination of human autopsy material is about as accurate as to attempt a reconstruction of the play of Hamlet from the final scene of the last act with the living and dead actors on the stage," so well summarizes the problems presented in a study of this complex disease process.

This work will continue to occupy an important place of ready accessibility in the surgeon's library.

GEORGE A. HIGGINS, M.D.

BOOKS RECEIVED

Books received are acknowledged in this section, and such acknowledgement must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers and as space permits.

The Postural Complex: Observations as to Cause, Diagnosis and Treatment. By LAURENCE JONES, B.S., M.D., Chief Orthopedist, Menorah Hospital, 1932–1943. Kansas City, Missouri. Visiting Orthopedist, Cedars of Lebanon Hospital, 1944–1953; Midway Hospital, 1948–1954, Los Angeles, California. Charles C Thomas, Publisher, Springfield, Illinois, 1955. Price $9.75.

The Mechanisms of Healing in Human Wounds: A Correlation of the Clinical and Tissue Factors Involved in the Healing of Human Surgical Wounds, Burns, Ulcers, and Donor Sites. By SHATTUCK W. HARTWELL, B.S., M.D., M.S., Ph.D. in Surgery, F.A.C.S., F.I.C.S., Diplomate of the American Board of Surgery, Attending Surgeon, Hackley Hospital and Chief of Surgery, Merch Hospital, Muskegon, Michigan; Surgeon, United States Army's 237th Station Hospital 1943–1944 (Utica, New York and New Guinea); Chief of Surgery, United States Army's 120th General Hospital, Manila, P. I., 1945; Member, New York Academy of Sciences. American Lecture Series #250. Charles C Thomas, Publisher, Springfield, Illinois. Price $4.75.

Counseling in Medical Genetics. By SHELDON C. REED, Director, Dight Institute for Human Genetics, The University of Minnesota. W. B. Saunders Company, Philadelphia and London, 1955. $4.00.

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Book Reviews, Classic Contributions to Surgery, Editorials and manuscripts on Surgical Technic are grouped together and are indexed under those headings in alphabetic order.